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## A Method of Roentgenologic Examination of the Shoulder<sup>1</sup>

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CALCIFICATIONS in the soft tissues are often seen in roentgenograms of the shoulder girdle. Fractures with separation of small cortical spicules of bone are not infrequent findings. Tendon injuries, as rupture of the supraspinatus tendon and dislocation of the long head of the biceps brachii, are also known to occur. On occasion, the roentgenologist may be called on to localize accurately such particles and other abnormalities. Stimulated by necessity, the authors early became aware that the usual roentgenograms of the shoulder, namely anteroposterior views with the hand in supination and pronation, are insufficient, even if taken in stereoscopic pairs. The great freedom of motion of the humeral head on the surface of the glenoid fossa actually adds to the difficulty of the exact localization of a lesion.

Anatomically the freedom of motion of the humerus is favored by a relatively loosely enveloping joint capsule, which attaches to the glenoid rim above and to the anatomical neck of the humerus below. This joint capsule extends in a tubular sheath enveloping the long head of the biceps brachii in its passage through the bicipital groove. Two layers of muscle overlie the shoulder girdle: the outer group, passing over the shoulder to insert into the upper humeral shaft,

consists of the deltoid, pectoralis major, latissimus dorsi, and teres major; the inner group, the so-called short rotators of the shoulder, include the supraspinatus, infraspinatus and teres minor inserting into the greater tuberosity, and the subscapularis, inserting into the lesser tuberosity. The subacromial bursa, somewhat smaller than the palm in a given individual, fits like a skullcap over the lateral aspect of the shoulder between these outer and inner muscle planes, obviously so located to prevent friction from contrasting movements (Codman, 1).

Because small calcifications so frequently appear within the short rotator tendons and periosteal proliferation may occur at the site of their insertion into the humerus, and because small bone fragments may actually be separated from these insertions, it is important to elaborate. The short rotator tendon insertions are broad and flat, measuring about an inch in length. They fuse into a continuous band or, better, interdigitate with one another. Fibers of this tendinous cuff are incorporated with the joint capsule proximal to their insertion. The greater tuberosity of the humerus forms an arc from before back, with its anterior margin bulging externally and sloping downward and forward for the insertion of the supra-

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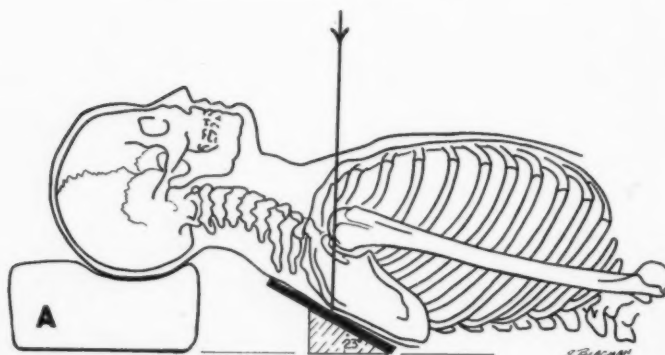


Fig. 1. A. First view: shoulder resting on 23°-angle board, the arm externally rotated, palm up.  
 B. Roentgenogram of shoulder in above position.  
 C. Roentgen skeletal study with lead foil placed on facets of the short rotator muscle tendon insertions. Lead strip *c* localizes the facet for the infraspinatus tendon. *a*, *b*, and *d* represent relative positions for the insertions of the subscapularis, supraspinatus, and teres minor muscles, respectively.

spinatus muscle; in the mid sector of this greater tuberosity a flat cortical plate of fingernail size slopes caudad externally and forms the facet for the infraspinatus insertion; and the less prominent posterior slope of the greater tuberosity marks the site for the insertion of the teres minor. The lesser tuberosity anteriorly (for the subscapularis) is a prominent "nubbin"-like projection. The intertubercular cleft

or bicipital groove divides the two tuberosities, and within it glides the long head of the biceps brachii to its point of insertion in the upper margin of the glenoid rim.

This study is on the order of that undertaken by Blackett and Healy (2), and their technic has been paralleled with some variation. Preliminary x-ray studies on the skeleton were made after placing lead foil over the individual sites of the inser-

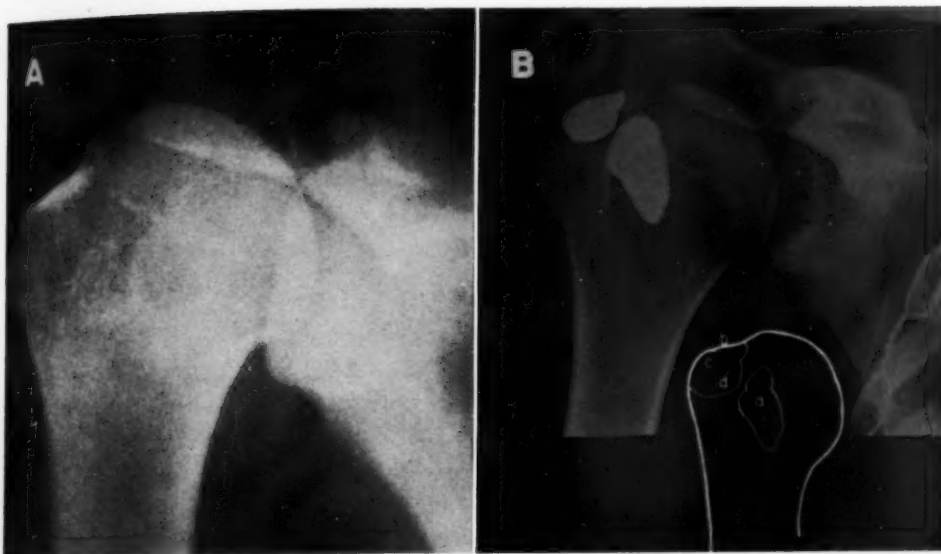


Fig. 2. A. Second view: position similar to Fig. 1, A except that angle board is removed.

B. Roentgen skeletal study. Lead strip *b* resting on the facet for the supraspinatus muscle tendon is profiled at its summit; *a*, *c*, and *d* represent facets for insertion of subscapularis, infraspinatus, and teres minor muscles, respectively.

tions of the short rotator tendons on the humeral tuberosities. These individual lead strips do not entirely represent the true picture of the short rotator tendon insertions, as some of their fibers extend beyond the limit of each facet, spreading over onto the adjoining facets and even upon the anatomical neck (sulcus). This tendinous cuff occupies about one-half the circumference of the anatomical neck with its attachments to both tuberosities (Fig. 5, D).

If, instead of individual pieces of lead, one lead strip were joined, a more nearly correct anatomical picture would be presented. Such a semicircular lead strip, however, could not be studied so easily, nor could the individual muscle insertions be identified.

The superior facet for the insertion of the supraspinatus tendon is curved anteriorly, so that the roentgenogram reflects in profile the tendinous fibers which have their point of insertion on its summit between the anterior and horizontal slopes. On occasion, a film with the central ray directed cephalad and tangentially may

be indicated (Fig. 6, A, ray A). By the same reasoning, more information may be obtained concerning the status of the infraspinatus tendon insertion. Likewise the subscapularis muscle tendon insertion may be examined in greater detail by rotating the arm slightly internally and externally from the original position (Fig. 4, Fig. 6, B, and Fig. 7).

The surgeon, at operation for removal of reported calcification, may be surprised to find more calcifications than the roentgenogram has demonstrated. Additional tangential views would have tended to bring out the true status more accurately.

Occasionally it becomes necessary to determine whether certain calcific deposits lie within a tendon or in the bursal sac. Since the bursa dips below the level of the facets on the lateral aspect of the greater tuberosity, calcifications which extend below the lowermost lead strip (insertion of the teres minor) can therefore be interpreted as in the bursa. Gravitational forces tend to carry these particles to the dependent portion of the subacromial bursa (Fig. 8, B and C).

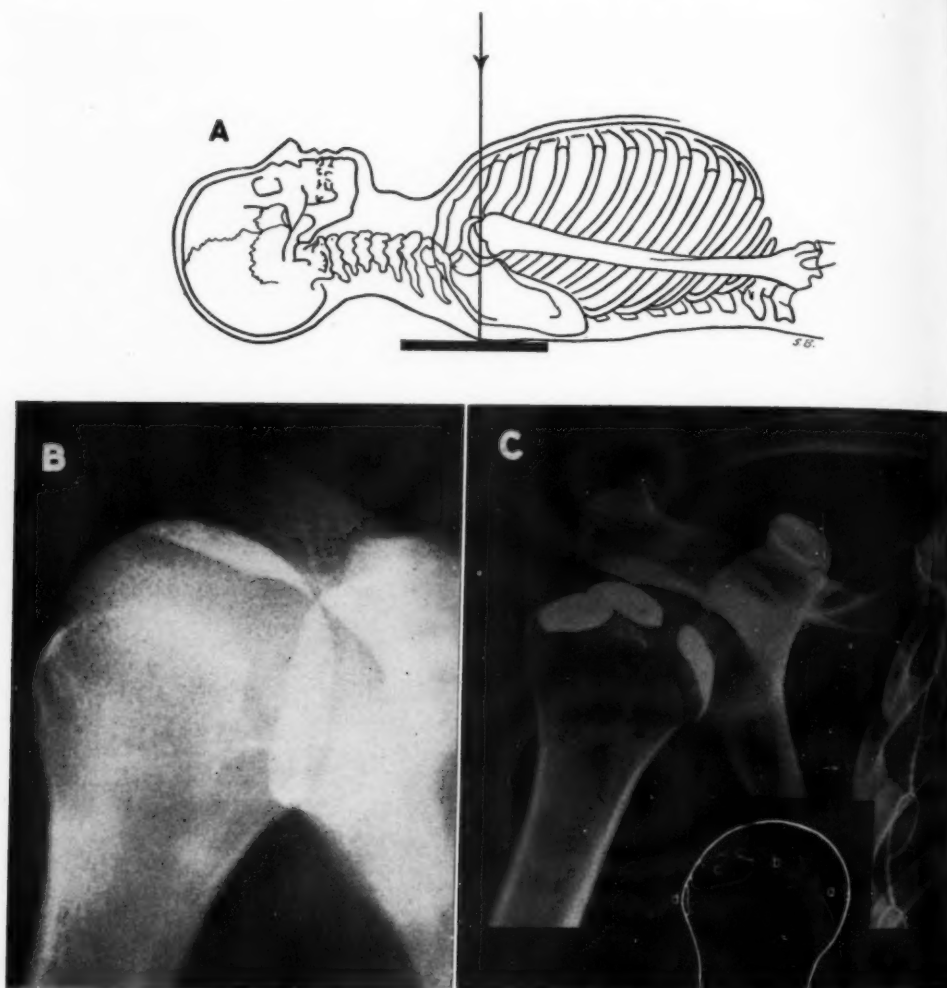


Fig. 3. A. Third view: patient in supine position, arm and forearm internally rotated, palm down.  
 B. Roentgenogram of shoulder with patient in above position.  
 C. Roentgen skeletal study demonstrating teres minor muscle insertion, *d*, in profile.

Care must be exercised in the inspection of roentgenograms of the shoulder, as many calcific deposits are minute and may contain but a small proportion of lime salt. A strong light will often bring into view such semi-opacities.

By such trial it was found that with some five projections the site of each muscle insertion could be brought into profile in at least two planes. Obviously each view should accurately localize any defect involving the periosteum and cortex and

any calcification or bone fragment in the adjacent soft tissues. The following views have been selected:

A. The first view is taken with the shoulder elevated on a 23-degree-angle board with the arm externally rotated and palm turned up. This view is designed to open the subacromial space between the humeral head and acromion of the scapula and bring into profile the tendon insertion of the infraspinatus muscle (Fig. 1, C-c). On occasion this view will bring calcifica-

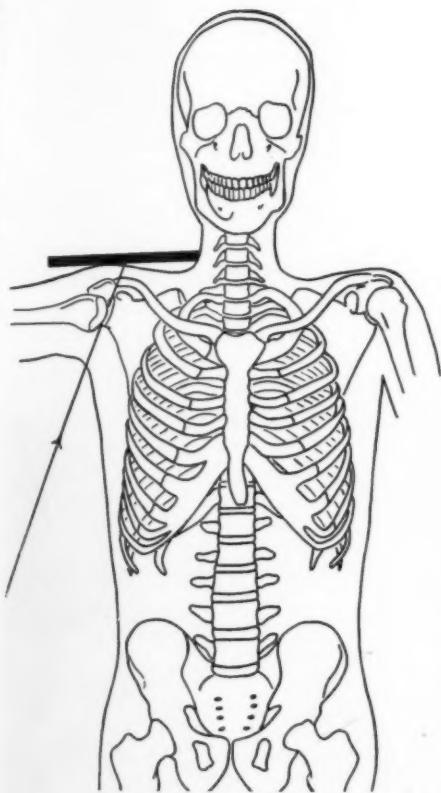


Fig. 4. A. Fourth view: Axillary view, patient seated, arm and forearm abducted to 90°, palm down. Central ray as illustrated. See also Fig. 4, B and C.

tions into view which might otherwise be overshadowed by the humeral head.

B. In the second view the angle board is removed and the arm and forearm remain externally rotated (Fig. 2). Thus the summit of the superior facet for the insertion of most of the tendinous fibers of the supraspinatus muscle on the greater tuberosity is seen in profile (Fig. 2, B-b).

C. In the third view, the patient remains in the supine position with the arm and forearm rotated internally; the palm is turned down on the table top (Fig. 3, A). This view is designed to rotate into profile the facet for the insertion of the teres minor tendon on the posterior aspect of the greater tuberosity (Fig. 3, C-d). Blackett and Healy (2) bring the teres minor tendon insertion into profile by placing the patient prone with the arm in internal rotation and the forearm flexed behind the back.

D. The fourth view, or axillary view (Fig. 4) is taken with the patient seated. The arm and forearm are abducted to 90 degrees from the patient's side; the internal and external epicondyles at the lower end of the humerus can be felt, and the plane of their axis is rotated to right angles with the floor. The central ray is angled medially and cephalad, *i.e.*, directed toward

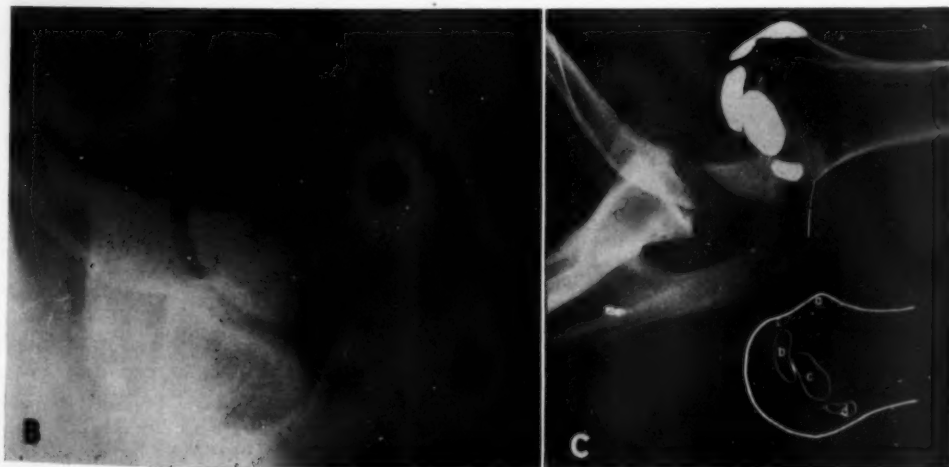


Fig. 4. B. Roentgenogram of shoulder with patient in above position. C. Roentgen skeletal study demonstrating subscapularis muscle tendon insertion on the lesser tuberosity, *a*, in partial profile.

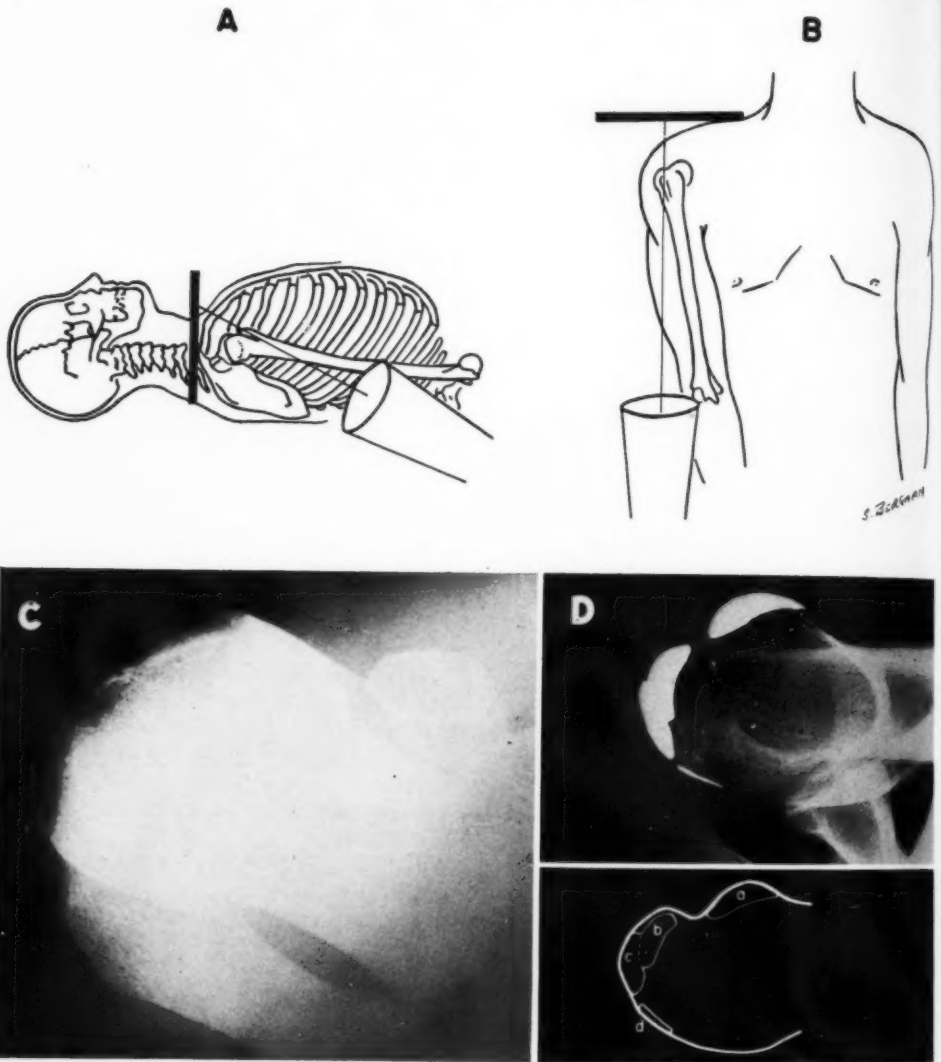


Fig. 5. A and B. Fifth view, bicipital groove; patient in supine position, arm externally rotated, palm up, elbow abducted close to body. Central ray 10-15° off the horizontal.

C. Roentgenogram of bicipital groove.

D. Roentgen skeletal study, *a* representing the insertion of the subscapularis muscle; *b*, insertion of the supraspinatus muscle. The bicipital groove between *a* and *b*, *c* and *d* represents the insertions of the infraspinatus and teres minor muscles. Note that the facet for the teres minor insertion is profiled.

the ear with the head erect. This brings the long axis of the acromial and coracoid processes into view along with the glenoid neck of the scapula. The gleno-humeral and acromioclavicular articulations are here visualized at right angles to the other projections. The surface of the lesser tuberos-

ity of the humerus has been rotated into profile anteriorly, demonstrating the insertion for the subscapularis tendon (Fig. 4, C-a, Fig. 9 and Fig. 11, A).

E. For the fifth view or so-called bicipital groove view (Sachs, Hill, and Chui-nard,<sup>3</sup>), the patient is in the supine position,

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his arm externally rotated to its fullest extent with the elbow held as close to the body as possible (Fig. 5, A and B). The tube is brought below the level of the table top, directed toward the head, angled upward by approximately 10-15 degrees (off the horizontal). The central ray is directed into the length of the bicipital groove. This view, besides giving an excellent delineation of the groove, also throws the tuberosities into relief with their osseous structures clear of the subjacent humeral shaft (Fig. 12, D). In addition, those tendon fibers inserted beyond the facets onto the body of the greater tubercle are demonstrable and frequently it is possible to confirm the exact location of calcifications visualized in the preceding views (Fig. 11, B).

Calcified deposits within the short rotator tendons are probably the end-result of trauma plus degeneration. Some old injury may have torn or shredded several tendon fibers, and incomplete healing or partial absorption of hemorrhage has resulted in calcium deposition. Such injuries of necessity do not have to include all the fibers of one tendon. Cortical bone fragments may also have been avulsed from the facet surface as a result of a so-called sprain fracture (Henry, 4). These calcific deposits arising from tendon injury may undergo natural resorption and disappear or enlarge and work their way to the superficial layers of the tendon adjacent to the floor of the subacromial bursa. While they are in this location and surrounded by products of degeneration, a sudden abnormal motion or effort, or even a normal one, may exert sufficient tension within the tendon to break the lining membrane and discharge the contents into the adjacent bursa (Codman, 1). Here they quickly diffuse throughout the bursa, irritating its delicate synovial surface and producing an acute bursitis. Calcified deposits in the tendons of the shoulder muscles do not of necessity cause pain; indeed, such deposits frequently may be discovered quite accidentally. These calcifications often occur bilaterally.

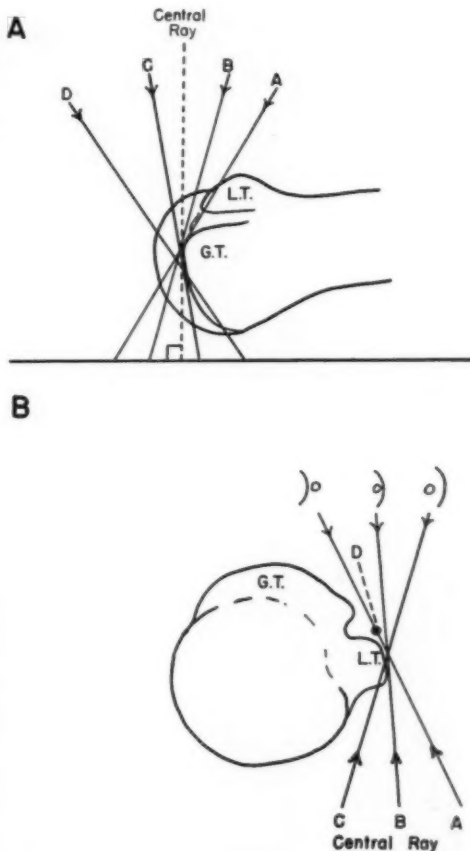


Fig. 6. A. Multitangential central rays: Dotted central ray indicates plane used for "profiling" the supraspinatus facet. Its anterior slope on the greater tuberosity may be profiled with central rays A and B. Similarly a view corresponding to ray C may be indicated to study the entire arc of the greater tuberosity.

B. Cross-section of humerus at level of lesser tuberosity (fourth view, Fig. 4). Routine central ray C fails to demonstrate calcification in subscapularis tendon at D. Additional views with central ray directed along plane A "clears" the deposit. Clearance may also be obtained by rotating the arm minimally. See Fig. 7.

According to Codman (1), roughening of the cortex with subjacent sclerotic changes at the tendon insertion in the region of the sulcus indicates previous tendon rupture or the existence of a low-grade chronic inflammatory process. Following complete or partial rupture of the supraspinatus muscle, an interruption in the surface continuity of the "cuff" develops and in the course of abduction of the arm permits the tuberosity to impinge

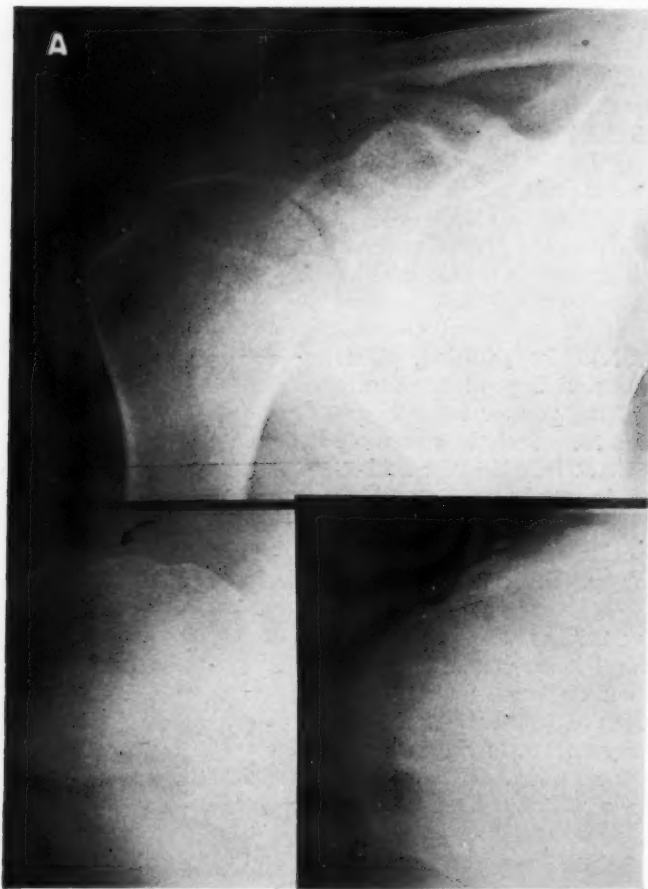


Fig. 7. Three views of same shoulder. A. Calcification overlying the greater tuberosity. B. Single calcification at the subscapularis muscle tendon insertion. C. Two calcifications seen overlying the lesser tuberosity. See Fig. 6, B.

upon the acromion, setting up a point of irritation with resulting bone proliferation on the tuberosity. The underlying bone frequently becomes absorbed, resulting in the formation of small cyst-like zones or caverns.

#### SUMMARY

A study of the roentgen anatomy of the shoulder has been made: (1) to locate more accurately the site of calcifications in the tendon sheaths of the short rotator muscles and subacromial bursa; (2) to clarify the exact location of fractures, particularly those involving the tuberos-

ities; (3) to identify and locate periosteal and subcortical zones of reaction secondary to tendon injury.

The usual routine views of the shoulder are insufficient. Five views are suggested:

A. Patient in supine position, hand in supination, shoulder elevated on a 23-degree-angle block to open up the articular space between the humeral head and the acromial process of the scapula and bring into profile the facet for the insertion of the infraspinatus tendon.

B. Patient in supine position, hand in supination, with the central ray at 180° to skirt the anterior margin of the greater

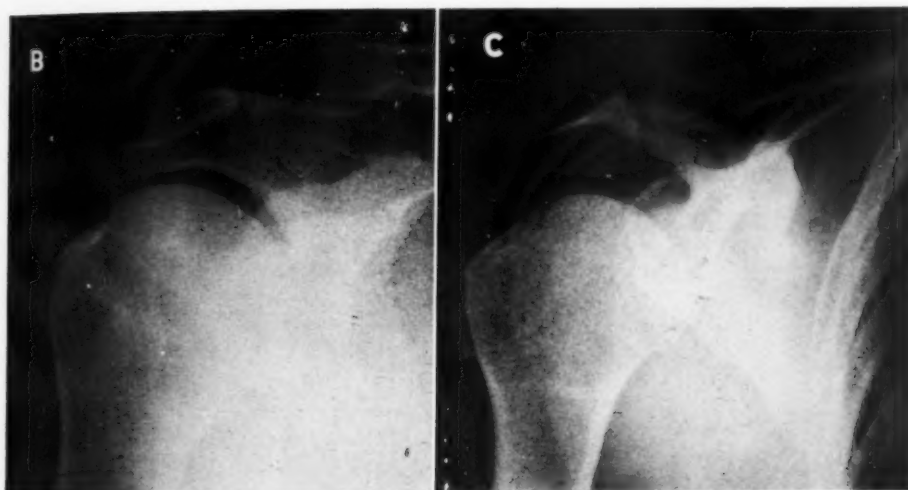
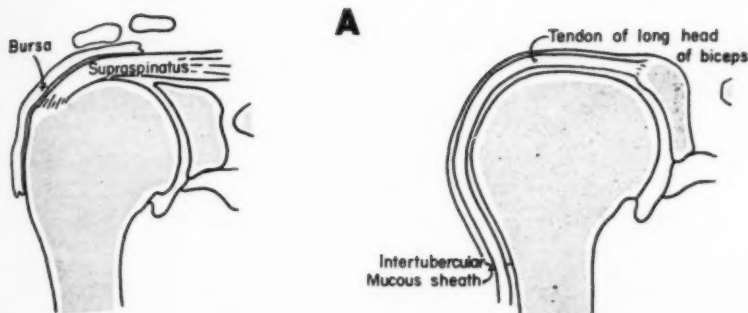


Fig. 8. A. Demonstrating the position of the subacromial bursa with relation to the supraspinatus muscle and tendon, acromion, and lateral aspect of the greater tuberosity.

B and C. Two different shoulders representing two stages in the process of extension of calcific deposits into the bursa from the supraspinatus tendon. In C the process is complete, with deposits reaching dependent parts of bursa.

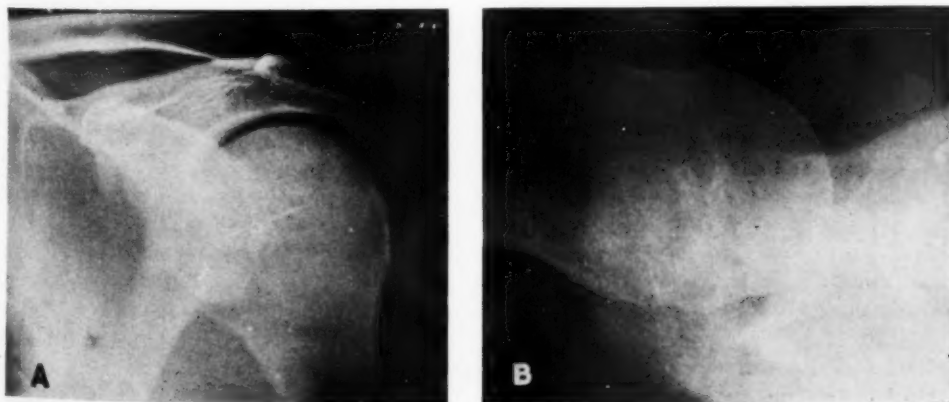


Fig. 9. A. Routine anteroposterior views were negative for fracture in previous examinations. B. Routine 4th view demonstrated a fracture of the tip of the coracoid process.

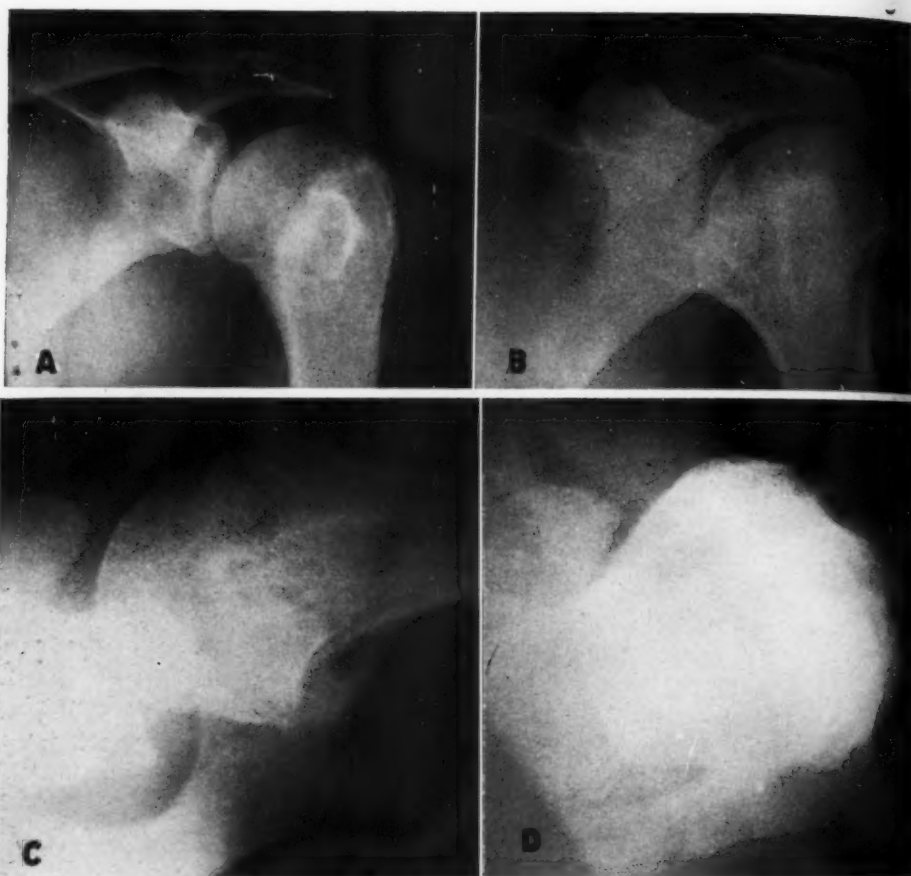


Fig. 10. Four views of a shoulder fifteen months after an oblique fracture through the surgical neck.  
 A. First view demonstrates irregular deformity of the greater tuberosity.  
 B. Third view demonstrates crescent-shaped deformity in the region of the teres minor.  
 C. Fourth view: union of fractured surgical neck with partial anterior displacement of the shaft fragment.  
 D. Fifth view shows encroachment of a fragment of the greater tuberosity on the bicipital groove.

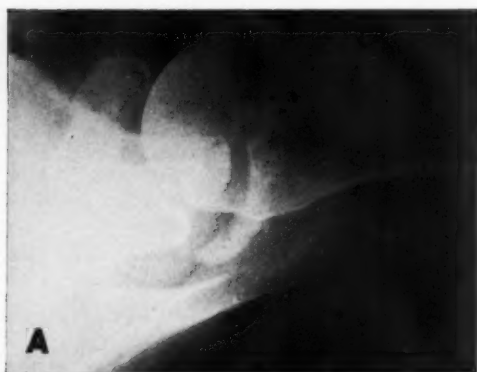


Fig. 11. Routine 4th and 5th views: axillary and bicipital groove views of the same shoulder. The crescent-shaped calcification corresponds to *d* in Figs. 4, C and 5, D and is therefore within the tendon of the teres minor muscles. Note additional calcification "picked up" in A at the insertion of the pectoralis minor muscle, on the coracoid process.

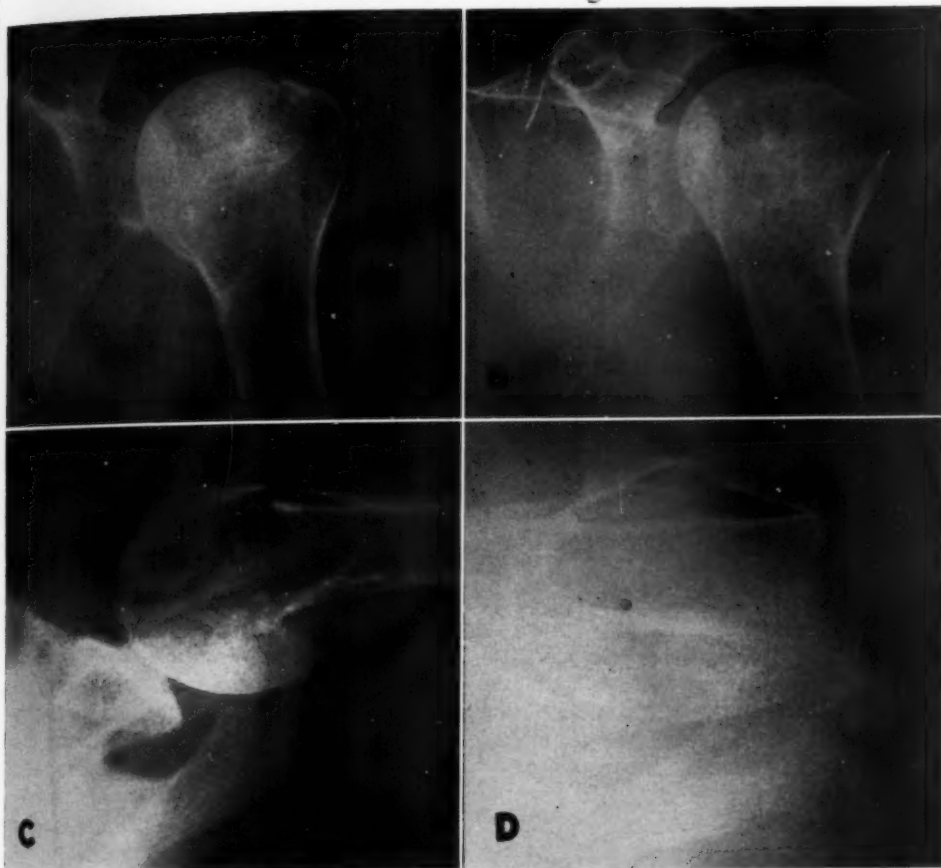


Fig. 12. A and B. Routine first and third views (external and internal rotation of the arm and forearm) reveal fracture of the surgical neck and greater tuberosity.

C. Axillary view demonstrates impaction into the humeral head posteriorly and anterior angulation.

D. Fifth view reveals the fracture of the greater tuberosity as gaping posteriorly.

tuberosity, to demonstrate the insertion of the supraspinatus tendon.

C. Patient in supine position, hand in pronation, with central ray at  $180^\circ$  to skirt the posterolateral margin of the greater tuberosity, bringing into profile the insertion of the teres minor tendon.

D. Patient in sitting position, with central ray directed upward and medially through the axilla, bringing out the glenoid neck as well as the two processes of the scapula and bringing into profile the lesser tuberosity, demonstrating the insertion of the subscapularis tendon anteriorly and the teres minor posteriorly.

E. Patient in supine position, hand in

exaggerated external rotation, tube below the table top and directed into the bicipital groove, bringing into profile the walls of the groove as well as both the greater and lesser tuberosities and further confirming localization of calcifications overlying either tuberosity.

Additional views may be taken at various degrees of angulation off these standards in specific instances.

Illustrations are shown demonstrating how various lesions can be accurately interpreted and localized by the technic described.

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## REFERENCES

1. CODMAN, E. A.: *The Shoulder*. Boston, 1934.
2. BLACKETT, CHAS. W., AND HEALY, THOMAS, R.: *Roentgen Studies of the Shoulder*. *Am. J. Roentgenol.* **37**: 760-766, June 1937.
3. SACHS, MAURICE D., HILL, HAROLD A., AND

CHUINARD, ELTON L.: *Further Studies of the Shoulder Joint with Special Reference to the Bicipital Groove*. *Radiology* **36**: 731-735, 1941.

4. HENRY, LUCAS S.: *Roentgenographic Evidence in the Tuberosity of the Humerus of Recent and Old Injuries to the Supraspinatus Tendon Attachment*. *Am. J. Roentgenol.* **33**: 486-490, 1935.

## SUMARIO

## Técnica para el Examen Roentgenológico de la Articulación del Hombro

Este estudio de la anatomía roentgenológica del hombro tuvo por objeto: (1) localizar mejor el asiento de las calcificaciones en las vainas tendinosas de los músculos rotatorios cortos y la bolsa sub-acromial; (2) esclarecer la situación exacta de las fracturas, y en particular de las que afectan las tuberosidades; y (3) identificar y localizar las zonas periósticas y subcorticales de reacción secundaria a lesión de los tendones.

Las vistas habituales del hombro resultan inadecuadas. Propónense las siguientes cinco vistas:

A. El enfermo en posición supina, con la mano en supinación y el hombro elevado sobre un calzo a un ángulo de 23 grados para entreabrir el espacio articular entre la cabeza del húmero y la apófisis acromial del omoplato y presentar de perfil la faceta para la inserción del tendón infraespinoso.

B. El enfermo en decúbito supino, con la mano en supinación, con el rayo central a 180° tocando el borde anterior de la tuberosidad mayor, para mostrar la inserción del tendón supraespinoso.

C. El enfermo en decúbito supino, con la mano en pronación, con el rayo central a 180° tocando el borde posterolateral de la tuberosidad mayor, perfilando la inserción del tendón del redondo menor.

D. El enfermo en posición sedente, con el rayo central asestado hacia arriba y el medio a través de la axila, poniendo de relieve el cuello de la cavidad glenoidea y las dos apófisis del omoplato y perfilando la tuberosidad menor, mostrando así la inserción del tendón subescapular por delante y del redondo menor por detrás.

E. El enfermo en decúbito supino, con la mano en rotación externa exagerada, con el tubo debajo de la cabeza de la mesa y asestado hacia el surco bicipital, perfilando las paredes de éste así como de las tuberosidades y confirmando además la localización de las calcificaciones sobrepuestas a una u otra tuberosidad.

En casos específicos, pueden tomarse otras radiografías a varios ángulos partiendo de estas pautas.

Los grabados demuestran la manera de interpretar y localizar exactamente varias lesiones con la técnica descrita.

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# Chronic Idiopathic Hypertrophic Osteo-Arthropathy<sup>1</sup>

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CHRONIC IDIOPATHIC hypertrophic osteoarthropathy is a condition occurring predominantly in males at the age of puberty or adolescence and characterized by the osteoarthropathic syndrome—clubbing of the digits, enlargement of bones and joints, and thickening of the skin of the face—in the absence of any demonstrable primary disease. Its course is slowly progressive to deformity and disability, which are often of extreme degree.

The nomenclature has been confounded by the dissimilarity of previous titles in the literature, such as familial acromegalooid osteosis (1, 2), idiopathic familial generalized osteophytosis (3), hypertrophic pulmonary osteoarthropathy without primary disease (4), and acropachydermia and pachyperiostitis (5). These terms have been used because of certain characteristics of the disease, namely, its familial tendency, superficial resemblance to acromegaly, similarity to other forms of osteoarthropathy, and the varying prominence of skin or skeletal changes.

The history of chronic idiopathic hypertrophic osteoarthropathy is naturally a part of that of the larger group of osteoarthropathic conditions. Hypertrophic pulmonary osteoarthropathy was the name coined by Marie (6) in 1890 in his classical paper to indicate an entity distinct from acromegaly, characterized by clubbed or Hippocratic fingers, arthritic symptoms, and bony thickening, that previously had been unrecognized. It soon became apparent that this syndrome was seen in association with a wide variety of

primary disorders, and cases began to appear, also, without evident primary disease. Sternberg (7) in 1899 pointed out that two of Marie's cases, those of the Hagner brothers, which had been sources of controversy since they were first reported twenty-five years previously (8, 9), appeared to constitute a separate group. However, it was not until Oehme (1) in 1919 reported his cases, together with the re-resurrected brothers Hagner, that the condition was recognized as a distinct entity. Oehme noted its relation to puberty and concluded that it was a constitutional disease made apparent by the endocrine imbalance of that period. In the dermatologic literature Grönberg (10) and others (11, 12) described similar cases in which there was marked thickening of the facies. As a rule, these reports attracted little attention, and even the most recent review of the general subject of osteoarthropathy (13), listing 337 references, merely mentioned that idiopathic types exist which are neither hereditary nor undiagnosed secondary types. The entire literature on the subject, except for three scattered reports (3, 5, 14), has appeared in foreign journals.

The incidence of the idiopathic form is at best low. In a review of the accessible literature, reports of 21 unquestionable cases of the disease have been found (1-5, 8-11, 14-25); 7 other probable cases (12, 26-30) could not be proved because of incomplete data. Four additional examples which have been encountered at the Mayo Clinic will be reported here, to-

<sup>1</sup> Abridgment of thesis submitted by Dr. Scanlan to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Radiology. Presented at the Thirty-third Annual Meeting of the Radiological Society of North America, Boston, Mass., Nov. 30-Dec. 5, 1947.



Fig. 1. Case 1. *a.* Left hand showing periosteal proliferation of the radius, ulna, and the bones of the hand with the exception of the carpals and distal phalanges. *b.* One and a half years later. Note progression of the process and normal closure of the epiphyseal lines. *c.* Forearm at the time the roentgenogram in *a* was made. Note the diaphyseal proliferation and the normal epiphyses and joint surfaces.

gether with a brief review and follow-up of one case previously recorded (14). The idiopathic form constitutes 3 to 5 per cent of all cases of osteo-arthropathy (13, 31).

The idiopathic condition is important despite its low incidence, since it represents a pure form of osteo-arthropathy untrammelled by the effects of primary disease and thus is ideal for study. The syndrome of osteo-arthropathy is in turn important not only because of its curious occurrence

in association with many widely dissimilar pathologic processes but also, probably from a more practical standpoint today, because of its relation to carcinoma of the lung. Osteo-arthropathy in primary pulmonary carcinoma has been seen in a severe, disabling form months and even years before attention has been attracted to the chest by respiratory symptoms; we have seen it on one occasion many months before the primary pulmonary lesion could be demonstrated by current

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methods. The discouragingly high incidence of advanced pulmonary carcinoma despite mass radiography, exploratory thoracotomy, and examination of sputum for malignant cells, scarcely condones apathy toward, or disinterest in, osteoarthropathy, a potential clue to earlier diagnosis, and should encourage the current interest in the physicochemical and immunologic diagnosis of cancer. Increased understanding of osteoarthropathy resulting from knowledge of the chronic idiopathic type may offer a new approach to the study of the mechanisms of disease and host reaction.

The prime purpose of this paper is to arouse interest in this little studied, potentially informative group of cases. It is hoped that knowledge of this facet of osteoarthropathy may eventually shed light not only on this abnormal bodily reaction but on other more significant reactions as well. Four typical examples and one atypical example will be presented. The condition is of particular roentgenologic interest because the skeletal manifestations are its most striking and ponderable aspect. Thus the roentgenologist is in the best position for diagnosis of the disease and elaboration of its mechanisms.

#### REPORT OF CASES

CASE 1: A male, 18 years of age, came to the Clinic complaining of enlargement of the hands and feet beginning at the age of sixteen. The condition was at first painless, but subsequently a painful swelling slowly developed on the distal portions of the extremities. This came on late in the day, was made worse by standing or exertion, and was relieved by rest and elevation of the extremities. The hands and feet became cold and blue on even mild exposure, and all the bones of the extremities had increased in size. In addition, there had been coarsening of the facial features and enlargement of the nose. Except for excessive sweating, increased fatigability and susceptibility to colds, the patient's general health was good at all times. He had to give up college and his part-time work as a steam fitter because of disability, and he became an accountant. Past and family history were non-contributory. A twin brother was said to be normal.

The patient was 5 feet, 7 1/2 inches (171 cm.) tall and weighed 145 pounds (66 kg.). He was a strongly built, red-haired youth, not appearing ill. The blood pressure was 140 systolic and 65 diastolic on

one occasion, and 134 and 76 on another. There were coarsening of the features, bulbous enlargement of the nose, and marked clubbing of the fingers. Other positive findings were hypertrophy of the nipples, feminine distribution of hair, acne vulgaris, and excessive perspiration of the hands, feet, and axillae. The extremities were strikingly enlarged distally with loss of muscle volume, thickening of the subcutaneous tissue, and non-pitting edema. There were rubor of the feet, cyanosis of the hands when they were dependent, and marked pallor of the hands when they were elevated.

Blood studies showed hemoglobin, 72 per cent; erythrocytes, 4,200,000; total cholesterol, 61 mg. per 100 c.c. of plasma; cholesterol esters, 31 mg.; lecithin, 61 mg.; fatty acids, 106 mg., and total fat, 167 mg. The basal metabolic rate was +13. The liver function test revealed grade 1 retention of bromsulfalein. No abnormality was noted in the urine, leukocyte and differential counts, Wassermann reaction, blood urea, creatinine, bilirubin, calcium, phosphorus, magnesium, potassium, sodium, and chlorides. Smears of the sputum were negative for acid-fast organisms. The reaction to the von Pirquet test was positive.

Roentgenograms of the chest, skull, spinal column, pelvis, and urinary tract were interpreted as normal. Epiphyseal development was normal. Roentgenograms of the extremities (Fig. 1) revealed thickening of the soft tissues and extensive subperiosteal proliferation of bone. The clavicles also were involved. Only the distal phalanges, the carpi, and tarsi showed no abnormal osteogenesis. The ungual tufts of the phalanges were absent. A fractured metacarpal healed in a normal manner.

Tissue obtained for microscopic examination revealed normal epidermis and increased fibrous tissue in the dermis and subcutaneous tissues, which also were thickened because of increased fat content. The periosteum (Fig. 2) had a thickened outer limiting layer and a very active osteogenetic layer. The vascularity was greatly increased, packing being required during biopsy of the fibula, because of bleeding. Striking hypertrophy of the media of the arteries was evident.

The patient was treated with parathyroid hormone for eight days without change and with a course of topical radium followed by five courses of low-voltage roentgen rays over a two-year period, with temporary improvement in the swelling. When last heard from, ten years later, he had moved to the southwest because he felt better in a hot, dry climate. He had married and had one child. There had been no remarkable progression of symptoms but they were still present and pressing him to the limit of his endurance.

The following case was reported in detail elsewhere (14) and only certain significant features and new follow-up data are given in this paper.

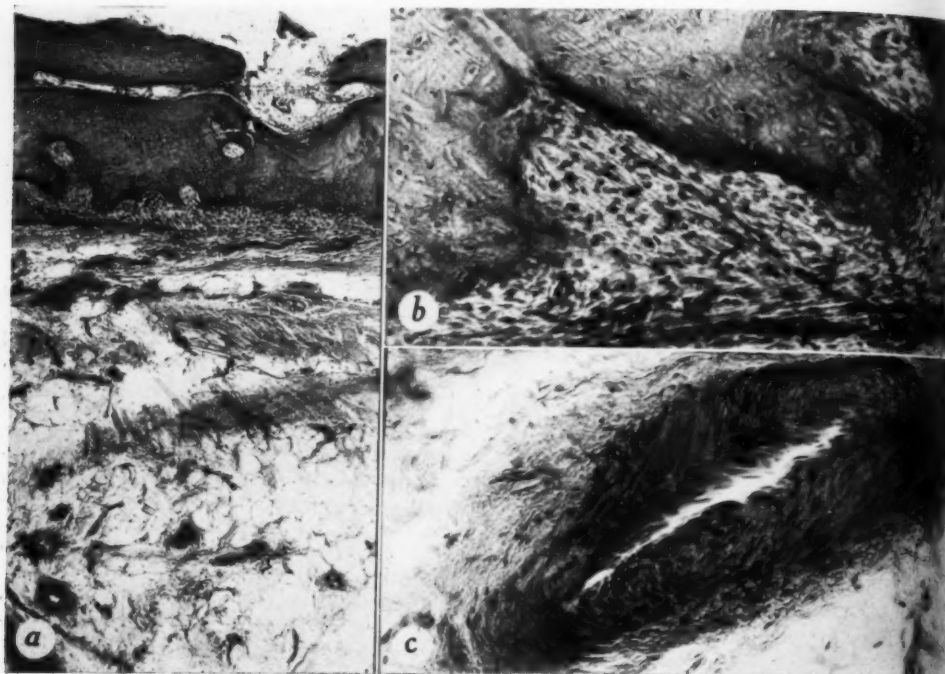


Fig. 2. Case 1. *a*. Section from tibia. Note thickened periosteum, inner and outer layers, fatty infiltration and hypertrophy of the arterial wall in lower left corner. Hematoxylin and eosin.  $\times 45$ . *b*. Higher magnification of section in *a*, showing the marked activity of the osteogenetic layer. Hematoxylin and eosin.  $\times 200$ . *c*. Tangential section of artery from same tissue, showing the massive medial hypertrophy. Hematoxylin and eosin.  $\times 200$ .

**CASE 2:** A 30-year-old white male, a rancher of Norwegian descent, came to the Clinic complaining of swollen, painful feet. He had noticed the onset of bony enlargement associated with excessive sweating at the age of thirteen. He was first examined at the clinic at the age of twenty-one and since that time had not noticed much change in the size of the bones. He had spent the nine months previous to his last admission in bed because of bouts of acute arthritis which had involved most of the joints of the upper and lower extremities at one time or another; the arthritis had finally settled in his feet. The joints remained stiff and sore between attacks; they were always better in the morning and worse at night, particularly after excessive use. The past history was non-contributory. The patient's maternal grandfather had had large, similarly shaped hands.

Physical examination revealed thickened features that produced a leonine appearance, clubbing of the fingers and toes, excessive perspiration, nodular mastitis of the left breast, and a mild kyphotic curvature of the thoracic part of the spinal column. The extremities were greatly enlarged distally, with large bulky joints and limited motion.

Laboratory examination revealed a hemoglobin value of 11.5 gm. per 100 c.c., a sedimentation rate of

57 mm. in one hour, and a value of 43 per cent for the oxygen saturation of the venous blood, with normal arterial saturation.

On roentgen examination, the gallbladder, stomach, small intestine, colon, skull, and chest were found to be normal. There was periosteal proliferation of marked degree involving the clavicles, scapulae, ribs, pelvis, and extremities (Fig. 3). The deposition on the clavicle and humerus was slightly more on the side of handedness. The long bones were roughened and, except for atrophy of the ungual tufts, the distal phalanges, carpi, and tarsi were normal. There was anterior wedging of several mid-dorsal vertebrae in the region of kyphosis, probably on a postural basis.

Except for some improvement in symptomatology and function as a result of physical therapy, the patient made little progress, and six months after dismissal he was still receiving disability compensation. However, he was able to do some work about his ranch when last heard from, nine years after the last visit to the clinic.

**CASE 3.** A 23-year-old white machinist of Greek descent had noticed gradual onset of swelling and enlargement of the distal part of the extremities at the age of fifteen, seven months after an appendec-

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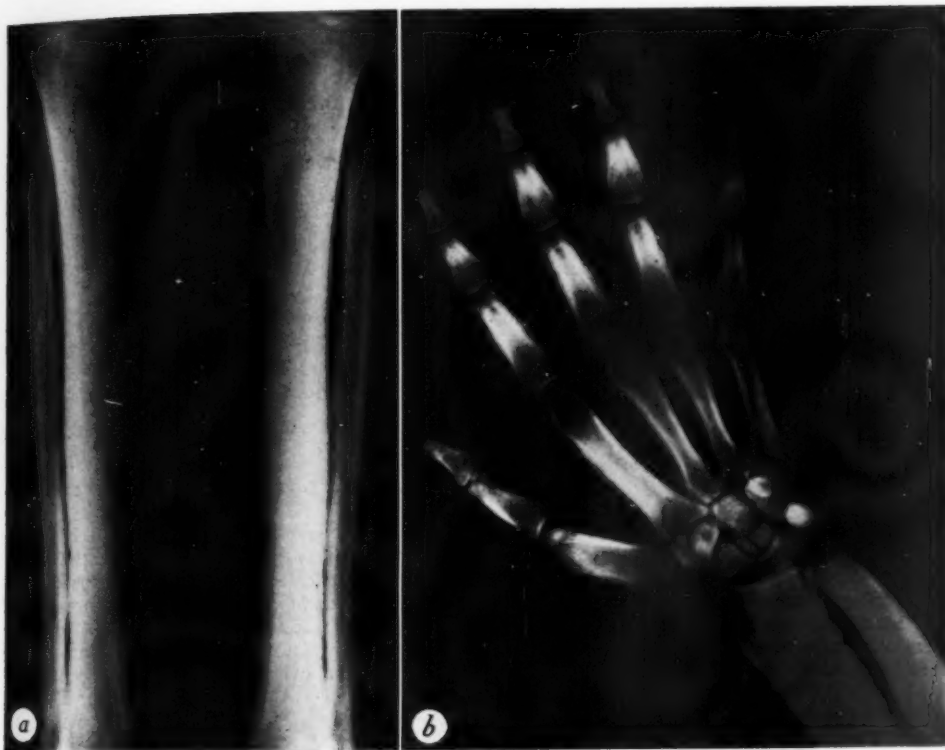


Fig. 3. Case 2. *a*. Thickening of the diaphyseal cortex of the long bones with alteration of the normal contour. Layering of the periosteal new bone is no longer evident. *b*. Similar changes are seen in all but the carpal bones and terminal phalanges. The latter show partial absorption of the ungual tufts.

tomy. This had progressed for eight years so that, although there had been no interference with activity, there was considerable deformity. Past history was non-contributory. No familial incidence was reported in parents or three siblings.

The patient was 5 feet, 8 inches (173 cm.) tall and weighed 146 pounds (66 kg.). His blood pressure was 110 systolic and 70 diastolic. He was well developed and well nourished. His features were thickened and the skin of the forehead was wrinkled. The distal extremities were markedly enlarged, with non-pitting edema, and there was clubbing of the digits. The enlargement was chiefly bony.

The blood sedimentation rate was 18. Nasal smears were negative for acid-fast bacilli. There was no abnormality of the urine, hemoglobin, or leukocyte and differential counts; the flocculation test for syphilis gave negative results. The basal metabolic rate, determined elsewhere four years previously, was  $-21$ , with a normal blood iodine content.

Roentgen examination of the chest was considered negative. The skull was reported normal. Roentgenograms of the extremities (Fig. 4) revealed marked soft-tissue thickening distally and extreme periosteal overgrowth of the long bones. The distal

phalanges of the thumbs showed proliferation anteriorly but were otherwise normal. Comparison with films made elsewhere four years previously revealed considerable progression.

A diagnosis of leprosy was considered as a remote possibility because of the leonine facies, but was later ruled out. The patient was dismissed and was not heard from subsequently.

**CASE 4:** An 18-year-old Scandinavian farmer complained of stiff and swollen joints. The onset had been abrupt—overnight—at the age of fourteen. The joints occasionally became swollen but the main complaint was the stiffness, which came on late in the day, particularly after the patient had been on his feet. He was much better in summer or on hot dry days than in winter or on cold damp days. His feet sweated excessively, and he believed this had caused an itching eruption that had been present for the past two years. The hands and feet were enlarged. The family, including six siblings, gave no history of similar involvement. The patient had been a blue baby at birth and had had rheumatic fever at four years of age. His health had otherwise been excellent until the present illness.

The patient was 5 feet, 11 inches (180 cm.) tall,

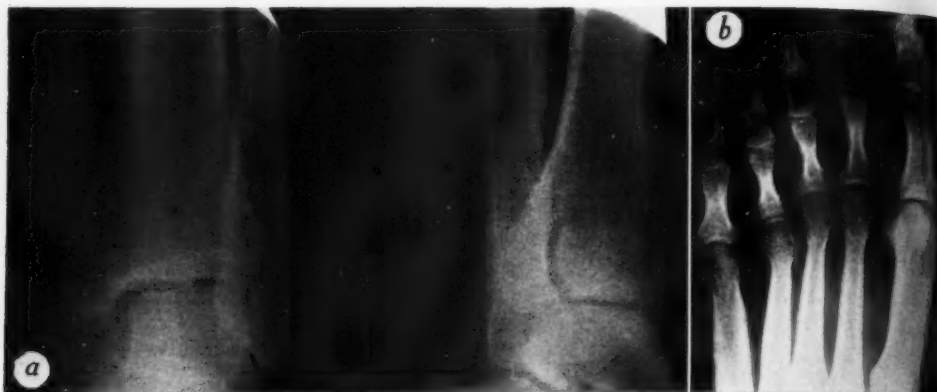


Fig. 4. Case 3. Periosteal proliferation of lesser degree involving (a) the metaphyses of the tibia and fibula and (b) the first and fifth metatarsals.

weighing 130 pounds (59 kg.). He was well developed and well nourished and had marked clubbing of the fingers and toes. The blood pressure was 128 systolic and 80 diastolic. The skin over the feet revealed a chronic eczematoid dermatitis. There was some diffuse lymphadenopathy of minor degree. The hands and feet were massively enlarged and there were some enlargement and reddening of the knee joints without significant limitation of motion. Atrophy of the quadriceps femoris was particularly marked. The heart was checked by several observers and nothing abnormal was noted.

Blood studies revealed a hemoglobin content of 13.1 gm. and normal leukocyte and differential counts. The urine was normal. The flocculation test for syphilis was negative. The values for uric acid, serum protein and cholesterol in the blood were normal. The electrocardiogram revealed no abnormality. The sedimentation rate was 16 mm. in one hour.

Roentgenoscopic examination of the heart was reported as giving negative results. Roentgenograms of the chest, including cardiac mensuration, and of the skull were interpreted as normal. The hands and knees were the only parts of the extremities roentgenographically examined. These showed some proliferation of bone in the region of the knee and striking absorption of the ungual tufts in the distal phalanges.

The patient was last heard from five years after dismissal. He had moved to Arizona, where he got along better in winter, but his condition was otherwise unchanged. He felt that his bones had not grown much since his last visit to the clinic.

#### The following case is an atypical one.

**CASE 5:** In 1917, a 37-year-old single woman was observed who had had a constant aching in her legs since childhood. This was always worse at night and was associated with tenderness. There had been

gradual enlargement of the extremities and, more recently, of the head. Four years previously, after an attack of pain behind the ear, a left facial paralysis had set in and had lasted for a month, without earache, headache, or malaise. The patient felt that there had been some recent loss of strength and that she was gradually failing.

The family history showed no similar involvement in parents or seven siblings. The past history was interesting in that the patient had led a semi-invalid life at home and that the menarche had occurred at twenty-one. Periods were normal.

The weight was 120 pounds (54 kg.). The height is not known. The systolic blood pressure was 116. The patient was a well nourished, poorly developed brunette appearing younger than the stated age. The digits appear clubbed in a photograph of the patient, although this point was not commented on at the time of examination. The features were coarsened and there was enlargement of the skull and grade 3 exophthalmos. The thyroid was not palpable. A left facial paralysis, of peripheral type, was present, and the edge of the spleen was barely palpable. The extremities were enlarged, shapeless cylinders, with large bones and scanty, swollen soft tissues. The muscles were atrophied and joint motions were limited.

Laboratory examination revealed a microscopic pyuria, a trace of albumin, a hemoglobin content of 68 per cent, 3,870,000 erythrocytes, a color index of 0.8, and slight anisopoikilocytosis in the smear. The basal metabolic rate was  $-1$ . Leukocyte and differential counts, blood and spinal fluid Wassermann reactions, and the Nonne-Apelt reaction were normal.

Available roentgenograms (Fig. 5) show massive enlargement of all the bones of the extremities, due to subperiosteal proliferation in the diaphyseal portions, to such an extreme degree that the bones were wider at mid-diaphysis than at their epiphyseal extremities. The carpal were uninvolved bilaterally,

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Fig. 5. Case 5. Massive periosteal proliferation involves, (a) both forearms and the metacarpals, with the exception of the fourth and fifth, (b) the tibia and fibula, and (c) the skull. d. Detailed view of the sella shows the mandible to be similarly involved.

as were the fourth and fifth metacarpals. The skull showed marked increase in bony width and density, most marked in the frontal bones, in the presence of a normal sella. The mandible was also quite markedly involved. Films of the chest were not available but had been reported as showing similar involvement of the ribs and clavicles.

In the absence of signs of hyperthyroidism other than the exophthalmos, the patient, after instruction on the use of symptomatic remedies, was dismissed as the subject of an unknown disease of the bones. When last heard from, seven years later, she was still disabled and miserable, and apparently her condition had undergone little change.

This case is considered atypical because of the early onset, slight clubbing associated with extreme bony change and, finally, normal fourth and fifth metacarpals of both hands.

#### SYMPTOMS AND SIGNS

From the foregoing histories and the literature it can be seen that these cases show a distinctive pattern. The combination of onset at puberty, slow progress, male predilection, familial tendency (60 per cent), and seasonal fluctuation is unlike any other type of osteo-arthropathy.

The symptoms are characteristic and follow a predictable pattern. The onset is insidious and the course slowly progressive, decelerating to a virtual standstill after many years. In some patients the changes are most prominent in the skin; in others, in the bones; and in still others, in the joints. Patients in whom the last-mentioned changes predominate show the greatest disability; those in whom the other changes are prominent show the greatest deformity.

General symptoms are surprisingly few. There may be some loss of energy or weakness and there are usually excessive sweating and vasolability. Secondary sexual disturbances, such as hypertrophy of male breasts, feminine distribution of hair, and scanty growth of beard, are common. However, libido and potency are unimpaired. Our female patient experienced a delayed menarche. Acne and seborrhea occur, as a rule. Fever is seen only with acute episodes of joint involvement. Exophthalmos was seen in one case.

Bony enlargement causes few symptoms. There is deformity, with a subjective sensation of heaviness, as a rule. One of our patients, the second to show involvement of the skull, had a facial paralysis, which may have been due to narrowing of the bony pathway of the nerve. Fractures heal normally and osteoporosis of disuse occurs in a normal manner.

Involvement of the joints may produce great disability. The usual symptoms

are enlargement, evening stiffness, and vague arthralgia. In certain cases these enlarged joints may give rise to real arthritis, with systemic reaction. Effusions are common and are sterile. Limitation of motion is the rule.

The skin changes may be striking. The diagnosis of leprosy is often suggested because of the thickened features. Large folds, resembling the gyri of the brain (*cutis verticis gyrata*) are seen, and in some cases plastic procedures have been done on the hypertrophied eyelids to enlarge the visual aperture. Similar changes are also seen in mentally deficient patients, in acromegaly, and in certain families in the absence of any other abnormality.

#### ROENTGENOLOGIC FEATURES

Roentgenograms are most important for an understanding of the osseous changes. Osteo-arthropathy is not likely to be confounded roentgenologically with any other disease. Roentgenographic examination should differentiate the types of osteo-arthropathy by qualitative and quantitative changes in the structure and by ruling out obscure primary disease. The bony change consists in accentuation of lateral growth by an unbridled proliferation of subperiosteal bone. This has been described in all bones but is best and most often seen in the long bones. Here proliferation is greatest over the diaphyses, is decreased over the metaphyses, and is slight to non-existent over the epiphyses. It is seen earliest and best in the distal third of both leg and forearm. The distal phalanges are unusual in that proliferation of bone does not occur or is minimal, and such changes as do occur appear only on the anterior surfaces. The ungual tufts as a rule undergo absorption or atrophy, although normal variations make this difficult to prove. Rarely is there demonstrable involvement of the carpus, tarsus, vertebrae, or skull. The endosteum does not participate in the hypertrophy, and the medullary cavity remains uncontracted. In one exceptional reported case (29) the bone was thickened, but endosteal re-

sorption kept pace with the periosteal proliferation so that the cortical width was normal.

#### LABORATORY AND PATHOLOGIC FEATURES

Laboratory studies have not, as yet, been a positive aid in idiopathic hypertrophic osteo-arthropathy. However, in a negative way they help to rule out other disorders. Routine studies reveal, as a rule, a mild hypochromic anemia and an acid urine. The blood chemical content is usually normal; however, a low value for lipoids in our first case and a low value for venous oxygen saturation in our second were observed. That further study should relate to metabolism and the endocrines is indicated, even without considering the clinical picture, by the frequent occurrence of unpredictable abnormalities of the basal metabolic rate, and by the reported lack of the specific dynamic reaction of protein and of reaction to injection of adrenalin, this lack usually being considered as evidence of pituitary abnormality (4, 10). Urinary gonadotropins were normal in one case (5). Circulatory dynamics should be studied, since they are the probable effector mechanisms of this disease. Bacteriologic studies have been fruitless.

The knowledge of the pathology is based on a single autopsy (9) and all too few biopsies. The fundamental change appears to be circumferential enlargement of the bone, hyperplasia of the connective tissue and fat in all layers, increased vascularity, and increase in the intercellular fluid. Clubbing of the nails is due to hypertrophy of both the nail bed and the nail curving over it. There is marked increase in the connective tissue of the corium of the skin. Joints show hypertrophy of the articular soft tissues, and biopsy of joints in 2 cases (2) in which severe arthritic symptoms and sterile, clear effusion occurred showed only mild round-cell infiltration. The bone is histologically and chemically normal except for a greatly thickened limiting membrane of the periosteum and a very active bone-forming layer. A most striking and

apparently quite constant finding is massive medial hypertrophy of the small peripheral arteries of the type seen in hypertension.

#### DIAGNOSIS, PROGNOSIS, AND TREATMENT

Diagnosis consists first of distinguishing osteo-arthropathy from other diseases and, second, of distinguishing its type. As a rule, the first task is not difficult, in view of clubbing, characteristic roentgenograms, etc. The second problem, however, may offer some real difficulty, since the osteo-arthropathic reaction appears to be basically the same in all types whether it involves one bone, one extremity, or all the bones. The type can be distinguished by considering the history, particularly as related to familial involvement, the age at onset, and the tempo and duration of the condition, and by carefully ruling out the presence of primary disease. Roentgenographic differentiation is reasonably accurate and is based on the same criteria. It will be considered later.

Prognosis as to life is good, but as to function, poor. Those who have predominant skin lesions seem to run the mildest course as regards disability, but the facial deformity is considerable. The patients who have primary involvement of bone get along fairly well until joint symptoms and swelling intervene, being on a midground as regards disability and deformity. The patients whose joint findings predominate are the most disabled and show the least deformity.

Treatment can be only symptomatic until the cause of the disease is understood. A change to a dry, hot climate, physical therapy, administration of analgesics, rest, and elevation of temperature are as yet the most effective measures. Plastic procedures have been necessary in certain cases of hypertrophy of the eyelid. A change to a sedentary indoor occupation is mandatory.

#### ETIOLOGY

The exact etiology of this condition is not known. The normal imbalance of the

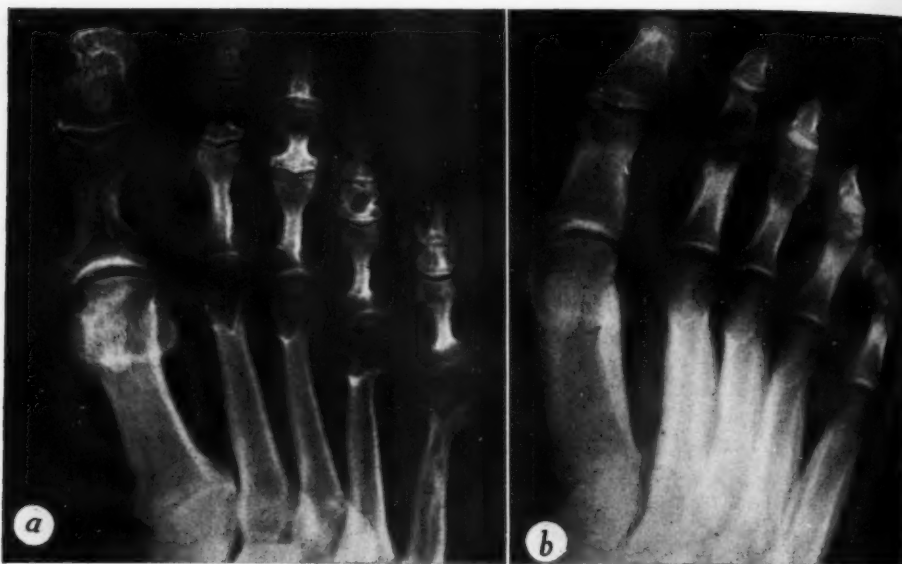


Fig. 6. *a*. The foot of a patient with acromegaly. *b*. The foot of the patient in Case 1. The unguis tufts, metaphyses, diaphyses, and general contour contrast sharply.

endocrines at puberty certainly appears to be at least the exciting cause. Hereditary predisposition is another factor, as evidenced by familial involvement in 60 per cent of the reported cases. Beyond this there is as yet only theory. For instance, one could hypothesize neuro-circulatory imbalance as the cause. The symmetrical, widespread changes, vasolability, and sweating would tend to incriminate a central mechanism. For example, the hypothalamus, through the production of abnormal stimuli, may be responsible for the medial hypertrophy of the arterial walls, thereby causing impaired nutrition, with the conversion of the peripheral tissues to a type whose nutritional requirements are less. Also, the pituitary has been suspect since the first cases were encountered, because it is the "master gland" whose influence at puberty is greatest, because abnormal secondary sexual characteristics are evident, and because other clinical and laboratory evidences of abnormal secretion of either the pituitary itself or of subordinate glands are observed. A constitutional defect based on genetic abnormality, with resultant

abnormalities of peripheral tissues, endocrines, metabolism, circulation, etc., could account for this condition. Obviously, only further detailed study of more cases with regard to endocrine, metabolic, and circulatory dynamics can account for the exact etiology.

#### ROENTGENOGRAPHIC DIFFERENTIAL DIAGNOSIS

Roentgenography is often of indispensable aid not only in distinguishing osteoarthropathy but in differentiation of the type. The diagnostic basis is the characteristic periosteal formation of new bone as related to distribution, tempo, and duration of the disease process, as manifested in the roentgenograms, coupled with the absence of evidence of disease in other parts of the body. The greatest difficulty is encountered in distinguishing the type of osteoarthropathy.

Of the systemic diseases capable of causing confusion, acromegaly has been, at least historically, the most important. Roentgenographically, acromegaly is almost the exact antithesis of chronic osteoarthropathy (Fig. 6). In acromegaly the



Fig. 7. *a.* Hypertrophic osteo-arthropathy secondary to a grade 4 (Broders) squamous-cell epithelioma of the right main bronchus. There had been symptoms of osteo-arthropathy for five months and respiratory symptoms for one month. *b.* More chronic form of hypertrophic osteo-arthropathy secondary to a malignant hemangioendothelioma of the lung. Osteo-arthropathic symptoms of two years' duration. No respiratory symptoms. Tumor discovered on routine examination of the chest and found inoperable at exploration.

short tubular bones are increased in length, as a rule with proportionately slender diaphyses and hypertrophied cancellous extremities. The ungual tufts are hypertrophic and the sites of tendon and ligamentous insertions are also hypertrophied. Degenerative joint changes are the rule in advanced cases, with resultant hypertrophic changes. All of these features are conspicuously absent in osteo-arthropathy, the opposite changes prevailing; namely, normal length and normal metaphyses, thickened diaphyses, atrophic ungual tufts, and normal epiphyses and joints.

Rheumatoid arthritis may be mistaken for chronic idiopathic osteo-arthropathy if the lack of morning stiffness of the joints and the presence of clubbing are over-

looked clinically. On roentgenograms only the early changes, occurring before periosteal proliferation is evident, might be confusing. However, by the time joint symptoms develop in osteo-arthropathy, periosteal proliferation of the distal long bones is already present and can be discerned, for instance, adjacent to the wrist in a film of a hand, or on the femur in a view of a knee. Periosteal proliferation, however slight, is the key to diagnosis.

Periostitis, specific and non-specific, offers difficulty only on single films. Syphilitic periostitis lacks the characteristic symmetrical distribution; for example, it occurs usually on tibias, not simultaneously on fibulas, and seldom on the bones of the forearm. It is usually not concentric. Non-specific periostitis, as for

example that associated with infections of the foot in diabetic patients, shows soft-tissue change and destruction of bone as well as the periosteal proliferation and, in addition, there is usually endosteal proliferation, with narrowing of the medullary canal.

Advanced osteitis deformans may be similar in some respects to advanced idiopathic osteo-arthropathy. The age of onset, presence of cystic changes or bowing deformities, distribution of involved bones, and disordered trabeculation with involvement of the medullary canal should lead to easy distinction. Osteopetrosis shows narrowing of the medullary canal and is thus readily distinguished.

Of the other types of osteo-arthropathy, the hereditary form may be difficult to differentiate after puberty (32). In this lifelong, mendelian dominant type of clubbing, the ungual tufts, as a rule, are hypertrophic. Periosteal proliferation is present on the distal long tubular bones but is never of extreme degree. The clubbed finger tips are prominent owing to the lack of soft-tissue thickening proximal to them. History of the hereditary trait may be needed for differentiation from cases of the early idiopathic type.

Secondary osteo-arthropathy is rarely difficult to distinguish and then only after puberty. Since the syndrome is most often acute in onset, is rapid in tempo, lasts months rather than years, and is associated with systemic disease, osteoporosis due to disuse accompanies the painful joints and is revealed in both the new and the old bone, the new bone usually being deposited as a single wide layer. The ungual tufts may or may not be absorbed (Fig. 7a). If the primary disease is chronic (Fig. 7b), slowly progressive, and of long duration, it may produce changes identical with those of the chronic idiopathic variety, as occurred in one case of congenital pulmonary arteriovenous aneurysm encountered at the clinic. In these instances, evidence of primary disease elsewhere may be discerned in other roentgenograms.

#### SUMMARY AND CONCLUSIONS

A syndrome of severe hypertrophic osteo-arthropathy not associated with primary disease and occurring predominantly in males at puberty has been presented, as observed in 5 cases at the Mayo Clinic and in 20 others reported in the literature. Although known as a distinct entity for twenty-six years, it has inexplicably attracted little interest, reports of only 3 of the 21 instances heretofore presented having appeared in the American literature. As this type of osteo-arthropathy is not associated with the effects of primary disease, it affords an excellent opportunity for the study and elaboration of mechanisms involved in this syndrome. Thorough exploration of the idiopathic type may well lead to ultimate clarification of these mechanisms. The abnormal growth of bone and the distinctive patterns should be of particular interest to roentgenologists.

A few conclusions may be drawn as regards the relationship of the chronic idiopathic type to other types of osteo-arthropathy. First, osteo-arthropathy can occur in the absence of primary disease or of mendelian dominant heredity. As a matter of fact, it can occur in its most extreme form in the absence of these conditions. Secondly, onset at puberty in these cases reveals close relation to the glands of internal secretion, particularly the pituitary. Thirdly, there would appear to be a central initiating factor, possibly pituitary or hypothalamic or both, and a peripheral effector mechanism, probably circulatory, and capable in certain instances of causing secondary clubbing and other characteristic changes in a single digit or extremity. The final factor and perhaps the greatest in importance is the constitutional predisposition which accounts for familial tendencies and variance in individual susceptibility to osteo-arthropathy.

#### REFERENCES

1. OEHME, C.: Familiäre akromegalieähnliche Erkrankung, besonders des Skelettes. *Deutsche med. Wchnschr.* 45: 207-209, 1919.
2. MÜLLER, WALTHER: Über die familiäre akromegalieähnliche Skeletterkrankung. *Beitr. z. klin. Chir.* 150: 616-628, 1930.

3. FREUND, ERNST: Idiopathic Familial Generalized Osteophytosis. *Am. J. Roentgenol.* **39**: 216-227, 1938.
4. BECKEN, SUSANNE: Zwei Fälle von Osteoarthropathie hypertrophante (P. Marie-Bamberger) ohne erkennbare Grundkrankheit. *Deutsch. Arch. f. klin. Med.* **187**: 117-131, 1941.
5. BRUGSCH, H. G.: Acropachyderma with Pachyperiostitis; Report of a Case. *Arch. Int. Med.* **68**: 687-700, 1941.
6. MARIE, P.: De l'ostéo-arthropathie hypertrophante pneumique. *Rev. de méd., Paris* **10**: 1-36, 1890.
7. STERNBERG, MAXIMILIAN: Acromegaly. Translation by F. R. B. Atkinson, London, New Sydenham Society, 1899.
8. FRIEDREICH, N.: Hyperostose des gesamten Skelettes. *Virchows Arch. f. path. Anat.* **43**: 83-87, 1868.
9. ARNOLD, JULIUS: Acromegalie, Pachyacrie oder Ostitis? Ein anatomischer Bericht über den Fall Hagner I. *Beitr. z. path. Anat. u. z. allg. Path.* **10**: 1-80, 1891.
10. GRÖNBERG, ALBERT: Is Cutis Verticis Gyrata a Symptom in an Endocrine Syndrome Which Has So Far Received Little Attention? *Acta med. Scandinav.* **67**: 24-42, 1927.
11. TOURAINE, A., SOLENTE, G. AND GOLÉ, L.: Un syndrome ostéo-dermopathique: la pachydermie plicaturée avec pachypériostose des extrémités. *Presse méd.* **43**: 1820-1824, 1935.
12. LABBÉ, MARCEL, AND RENAULT, PAUL: L'ostéodermpathie hypertrophante. *Presse méd.* **1**: 545-546, 1928.
13. MENDLOWITZ, MILTON: Clubbing and Hypertrophic Osteoarthropathy. *Medicine* **21**: 269-306, 1942.
14. CAMPBELL, D. C., SACASA, C. F., AND CAMP, J. D.: Chronic Hypertrophic Osteoarthropathy. *Proc. Staff Meet., Mayo Clin.* **13**: 708-713, 1938.
15. HOFFMANN, MAX: Bemerkungen zu einem Fall von Akromegalie. *Deutsche med. Wchnschr.* **1**: 383-385, 1895.
16. SISSON, R. J.: Cutis Verticis Gyrata. *J. A. M. A.* **86**: 1126-1127, 1926.
17. RENANDER, AXEL: Skelettveränderungen bei einem Fall von Cutis verticis gyrata. *Acta radiol.* **9**: 399-409, 1928.
18. ISRAELSKI, MARTIN, AND POLLACK, HERBERT: Beitrag zur Osteoarthropathie hypertrophante nach Pierre Marie beziehungsweise toxischen Osteoperiostitis ossificans nach Sternberg. *Röntgenpraxis* **2**: 342-352, 1930.
19. RENANDER, AXEL: Cutis verticis gyrata-Akromegalie-Osteoperiostitis hyperplastica. *Acta radiol.* **18**: 652-669, 1937.
20. RENANDER, AXEL: Hypophysenbestrahlung bei Cutis verticis gyrata. *Acta radiol.* **19**: 254-258, 1938.
21. STEPHAN, ERNST: Über die "Osteoarthropathie hypertrophante pneumique" (Pierre Marie-Bamberger). *Deutsch. Arch. f. klin. Med.* **182**: 183-192, 1938.
22. BENÁRD, MIKLÓS, AND KOVÁCS, ÁKOS: Osteoarthropathie hypertrophante mit Veränderung des endokrinen Systems. Zur Genese des Syndroms Marie-Bamberger. *Fortschr. a. d. Geb. d. Röntgenstrahlen* **62**: 316-321, 1940.
23. CARRUTHERS, L. B.: Idiopathic Hypertrophic Osteoarthropathy Familial in Type. *J. Christian M. A.* **18**: 1-3, 1943.
24. SYMONS, T. H.: Case of Pulmonary Osteoarthropathy. *Indian M. Gaz.* **39**: 16-18, 1904.
25. APERT AND BIGOT: Ostéo-arthropathie hypertrophante (type de P. Marie). *Bull. et mém. Soc. méd. d'hôp. de Paris* **2**: 1715-1717, 1921.
26. VAN DER WEIJDE, A. J. AND BOEKHOUDT, H. B.: Een geval van osteoarthropathie hypertrophante. *Nederl. tijdschr. v. geneesk.* **2**: 781-787, 1895.
27. GLUZINSKI, A.: Quoted by Locke, E. A. (31).
28. RAMOND, LOUIS, AND BASCOURRET, MAURICE: Un cas d'ostéoarthropathie hypertrophante de Pierre Marie. *Bull. et mém. Soc. méd. d'hôp. de Paris* **50**: 1015-1023, 1926.
29. MANKOWSKY, B. N., HEINISMANN, J. I., AND CZERNY, L. I.: Osteopathia dysplastica familiaris. (Zur Genese des Syndroms Marie-Bamberger.) *Fortschr. a. d. Geb. d. Röntgenstrahlen* **50**: 542-549, 1934.
30. ROY, J. N.: A propos de la pachydermie plicaturée avec pachypériostose des extrémités. *Presse méd.* **45**: 403-404, 1937.
31. LOCKE, E. A.: Secondary Hypertrophic Osteoarthropathy and Its Relation to Simple Club-Fingers. *Arch. Int. Med.* **15**: 659-713, 1915.
32. WITHERSPOON, J. T.: Congenital and Familial Clubbing of the Fingers and Toes, with a Possibly Inherited Tendency. *Arch. Int. Med.* **57**: 18-31, 1936.

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#### DISCUSSION

**Donald S. Childs, M.D.** (Syracuse, N. Y.): First, as to the paper by Dr. Howes and Dr. Alicandri: I was interested to note the definite anatomical structures and relationships in profile views that were shown for the insertions of the three facets on the greater tubercle of the humerus. I was struck also by the fact that in Brooklyn they apparently can get their patients in a much better position than we can in up-state New York. We can't get our patients with shoulder injuries to abduct and rotate their arms. Sometimes we feel that we get a considerable amount of additional information from a direct lateral view of the shoulder (as suggested by Dr. Wm. G. Herrman of Asbury Park, N. J.)—not a lateral view of the chest, but a direct lateral view of the shoulder with the central ray coming just beneath the scapula, with the film at right angles. This will show the coracoid process and also the glenoid and the head of the humerus.

Dr. Camp and Dr. Scanlan in their paper on Idiopathic Hypertrophic Osteo-Arthropathy have offered us a challenge and we should take it up. We see clubbed fingers, with no apparent disease in the chest; there is a possible suggestion that this may be an early indication of carcinoma of the lung. We noticed that the bone is elongated, that it is dense, that the ungual tufts are not enlarged, and that the bone is of bone, not laid down on bone and not from bone. The essayists' experience is a challenge to us to watch for more of these cases.

I was interested also in the skin changes and their resemblance to leprosy. This feature was observed, I believe, in only one of the cases presented, but thickening of the skin of the face, thickening of the bones, and clubbing of the fingers were mentioned as indicative of the idiopathic hypertrophic osteoarthropathic syndrome. There was no change at all in the sella.

## SUMARIO

## Osteoartropatía Hipertrófica Idiopática Crónica

A base de 5 casos observados en la Clínica Mayo y de otros 20 que aparecen en la literatura, describese un síndrome de osteoartropatía hipertrófica grave que no se asocia con enfermedad primaria y predomina en los varones en la pubertad. Aunque reconocido como entidad bien definida desde hace 26 años, por alguna razón inexplicable ha llamado poco la atención, y sólo 3 de los 21 casos comunicados hasta ahora corresponden a los Estados Unidos. Como esta forma de osteoartropatía no se vincula con los efectos de una afección primaria, ofrece una magnífica oportunidad para el estudio y exposición de los mecanismos que intervienen en este síndrome. La detenida exploración de la forma idiopática puede muy bien conducir al fin al esclarecimiento de dichos mecanismos. La hipertrófia ósea y los característicos moldes que toma deben revestir interés especial para el radiólogo.

Cabe sacar algunas conclusiones con respecto a la relación de la forma idiopática con otras osteoartropatías. En primer lugar, esta artropatía puede presentarse en ausencia de enfermedad primaria o de herencia dominante mendeliana, y en efecto puede revestir la forma más grave a falta de esos factores. Segundo, su iniciación en la pubertad señala una íntima relación con las glándulas endocrinas, y en particular la hipófisis. Parece que debe haber un factor iniciador central, posiblemente hipofisario o hipotalámico o ambos, y un mecanismo actuador periférico, probablemente circulatorio, capaz en ciertos casos de provocar deformación secundaria y otras alteraciones típicas en un solo dedo o miembro en ausencia de un factor central estimulante. El factor definitivo, y quizás el más importante, es la predisposición orgánica que explica las tendencias familiares y la variada susceptibilidad.



## The Roentgen Appearance of Ossifying Fibroma of Bone<sup>1</sup>

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ACCORDING TO Eden (2), the disease generally known as ossifying fibroma was first described in 1872 by Menzel, who considered it to be a form of osteoma. At least two early textbooks on the jaw, that of Heath in 1894 (5) and of Scudder in 1912 (12), contain accounts of tumors consistent with the lesion under discussion here. Montgomery (8) in 1927 probably introduced the term ossifying fibroma. At this time he described 3 cases and in a review of the literature was able to uncover 14 similar ones. Geschickter's paper (4) on jaw tumors in 1935 contained a section on ossifying fibroma. Two years later Phemister and Grimson (9) in a significant study presented 13 cases, reviewed 2 of Montgomery's, and noted 30 more from the literature. These authors, employing the term fibrous osteoma for ossifying fibroma, suggested that this process bore a relationship to membranous bone similar to that which osteochondroma bears to cartilaginous bone. In 1939 Eden presented a study of 5 cases primarily from the pathological point of view and concluded that ossifying fibroma represents an immature form of the benign "fibro-osseous" tumors of membranous bone.

Following the presentation of fibrous dysplasia of bone as an entity, several writers have suggested an intimate relationship between this disease and ossifying fibroma. In fact, Lichtenstein (6) felt that several of the cases of Phemister and Grimson were indistinguishable from fibrous dysplasia. Four years later, in 1942, Mallory (7) seemed ready to accept the possibility of these diseases being one and the same. Schlumberger (11), writing in 1946 and basing his opinion largely upon pathological grounds, concluded that os-

sifying fibroma was at the most a variant of fibrous dysplasia.

It is the feeling of the Department of Pathology at Memorial Hospital that ossifying fibroma of the jaw presents little of a specific nature histologically. Consequently the department refrains from making an unequivocal diagnosis upon microscopic study alone. In material seen at this hospital, however, there are a few tumors in the group under discussion which are outstanding microscopically in that they show relatively extensive calcific spherulation. This has not been observed in sections of any of our cases having the clinical, radiographic, and pathologic attributes of fibrous dysplasia of bone. Although this cannot be considered an absolute differential criterion, it can scarcely be entirely ignored (3).

Generally speaking, when histologic material is adequate, one readily places ossifying fibroma in a small group having certain similar microscopic features. This group includes fibrous dysplasia of bone as well as fibrosing epulis and osteitis fibrosa-cystica. The segregation of ossifying fibroma depends upon its location, either the mandible or the maxilla being the usual site; upon evidence that the lesion is from or arises within bone; at times upon the clinical course of the disease and blood chemistry findings; and finally, as the studies to be reported here have led us to believe, upon the radiographic appearance.

We consider ossifying fibroma of bone to be essentially a monostotic process, limited in occurrence to the membranous bones and rarely seen except in the mandible or maxilla. It possesses many of the features of a benign tumor. It occurs typically in childhood, being discovered as a pain-

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less swelling of the face. If untreated, this swelling slowly enlarges over a period of months or years, showing, as it ages, a tendency to the deposition of calcium within it. There seems to be evidence of a slowing of the growth rate later in life. The process is always monolocular, having a thin bony capsule about most of the tumor, and, particularly as it grows in the maxilla, possessing the property of destroying nearby bones without displacing them significantly. On gross pathologic examination, the tumor is seen to have a thin calcified shell about 1 mm. thick and a delicate fibrous capsule. The cut surface of the lesion is firm, white to gray, sometimes moist and glistening. A scattering of calcified areas is present, often in the form of spicules or spherules.

In the light of the above findings, it seems to us that ossifying fibroma of bone presents sufficient uniqueness to warrant its being considered a separate disease.

We have had the opportunity of studying from a radiological standpoint 12 ossifying fibromas of bone in which the pathological and clinical material was adequate. The follow-up period averaged about five years, calculated from completion of the initial treatment. The shortest follow-up interval was several months and the longest thirteen years. The material was selected from a slightly greater number of related jaw lesions, a few of which we have been unable to classify satisfactorily in the light of present knowledge. We feel that there is sufficient correlation within this group of 12 tumors to make later shifting of material of any significant degree unlikely. Dr. Frank W. Foote, Department of Pathology, Memorial Hospital, kindly reviewed the slides on these cases.

It is the purpose of this paper to present the roentgen features of ossifying fibroma, together with significant aspects of the clinical course and treatment as they affect roentgen diagnosis.

#### ROENTGENOGRAPHIC FEATURES

In the following discussion of the roentgen appearance of ossifying fibroma of

bone, the findings described are those in the earliest radiographs available in each instance. The roentgen picture as given is thus of the early uncomplicated growth. Later, an evaluation of the effects of radiation, curettage, and growth will be made. Here the roentgen appearance of this disease is resolved into what we consider to be the essential basic components.

*Bone Involved:* In 5 cases the ossifying fibroma occupied some portion of the mandible, while the maxilla was the site of 6. In Case 4 the tumor was located in the temporal bone, apparently arising from the tympanic part, which is of membranous origin. Generally speaking this ossifying fibroma of the temporal bone was found to resemble the maxillary tumors. Sites of multiple origin have occasionally been noted, a single patient having a tumor in the maxilla and one in the mandible. The tumors in this series were all monostotic. The fact that ossifying fibroma has almost invariably been reported as arising either from mandible or maxilla seems to be a significant point in its diagnosis.

*Location in Bone:* The tumors of the mandible were located chiefly in the medullary area and seemed to arise from a medullary source. There was no predilection for any particular segment of the bone.

It was generally impossible to postulate the origin of the maxillary tumors, because of the thinness of the bone segment involved. In one case (Case 8) it seemed that the tumor might have begun subperiosteally, since the process was seen to rest upon the surface of unaffected bone. A similar type of origin could be suspected in several other maxillary cases.

*Size of Lesion:* Each tumor was measured on the earliest roentgenogram. In a few cases there was a delay of several months from the initial symptom to the time of the first roentgen study. In spite of this, it is believed that the dimensions recorded from x-ray measurement give a fairly accurate estimate of the size at the onset of symptoms. It seemed apparent

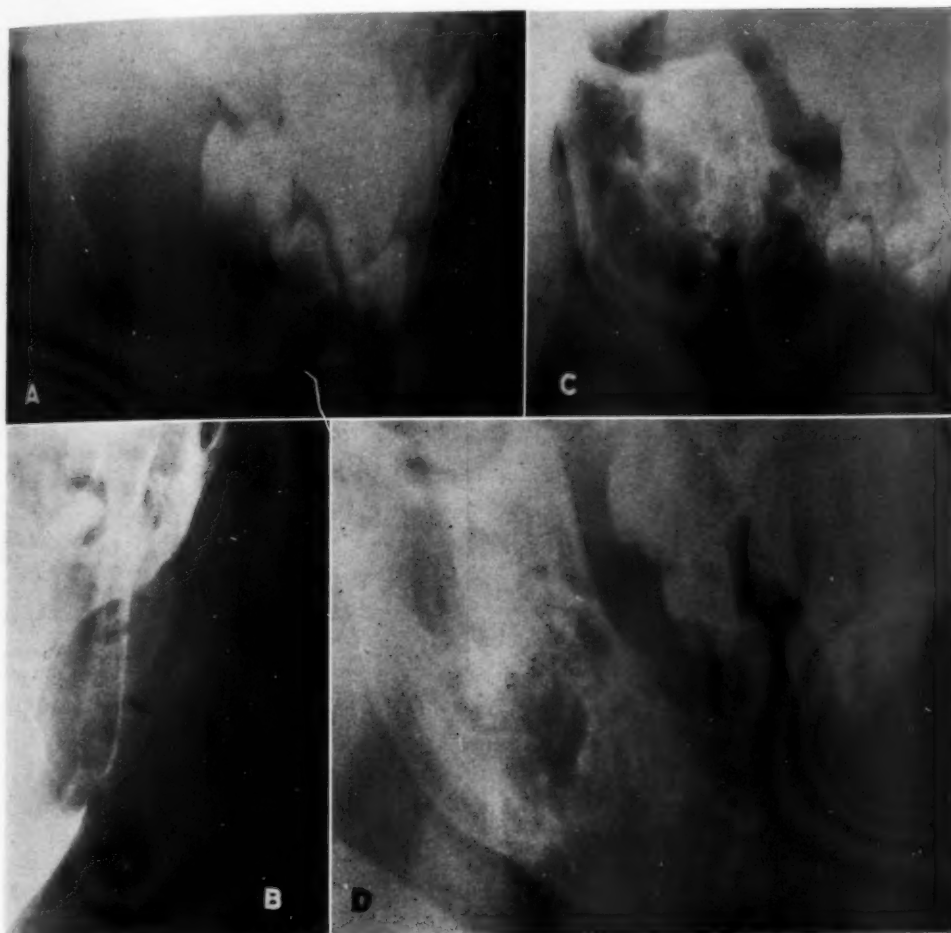


Fig. 1. Case 1. A. Ossifying fibroma seen as a unilocular, structureless lesion displacing adjacent tooth. B. Sagittal view showing egg-shell margin of tumor. C. Evidence of recurrence ten months after surgical treatment. D. Five years after x-ray treatment for the recurrence there is some improvement in configuration of the mandible with increased density and no evidence of active tumor.

that the location of the tumor in a readily visible area was a more important cause for early discovery than was size alone. The smallest tumor measured  $3 \times 2.5 \times 2.5$  cm. and occurred in the maxilla. The largest was a mandibular tumor,  $8 \times 6 \times 6$  cm. The average size at the time of admission was  $5 \times 4 \times 3$  cm.

**Configuration:** The shape of ossifying fibroma was regularly found to be either oval or approaching the spherical. This was one of the more constant findings and to it considerable significance is attached.

**Bone Destruction and Production:** With

a single exception (Case 11), all of the tumors in this series were predominantly destructive, and the overall density was distinctly less than that of the normal neighboring bone. The exception was a mixed type of lesion, with a somewhat greater amount of calcific density, making it slightly more radiopaque than normal bone. It seems significant that this tumor was a relatively old one when first demonstrated radiographically. It is our impression that, as these growths become older, there is a tendency toward greater density due to the formation of small



Fig. 2. Case 2. Ossifying fibroma, demonstrable as a unilocular, expansile process with egg-shell margin and a few vague calcified striae.

calcific areas which still later tend to converge. The only productive change in the earlier lesions was the occasional presence of a few delicate striae, partial septa, or small scattered calcific flecks. It did not appear that location, whether maxillary or mandibular, had any particular bearing upon the degree of destruction or production.

In short, we have found ossifying fibroma to be a predominantly destructive tumor when seen early, with a tendency later to show increased productive changes which seldom become great enough to overtake the earlier destructive ones.

*Direction of Growth:* When the tumor originated from the medullary cavity, the growth tended to remain within the bone, expanding equally in all directions. If the lesion began on the surface of the bone, *i.e.*, subperiosteally, it tended to grow in this region destroying adjacent bone and extending outwards to equal degrees, still preserving a capsule. The oval or spherical configuration suggests that growth is outwards about equally in all directions from a central point of origin.

*Periosteal Reaction:* In none of the tumors in this series was there evidence of a periosteal reaction:

*Effect on Adjacent Structures:* In discussing the effects upon neighboring areas,

consideration will be given first to the growth as it appears in the mandible and then as it is seen in the maxilla.

In the mandible the tooth is the organ most often affected. All 5 mandibular cases showed some degree of tooth displacement. At times this was so slight as to escape detection by the patient.

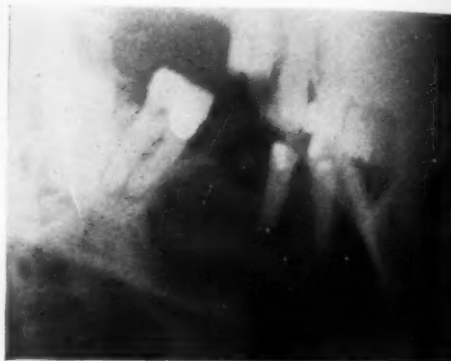


Fig. 3. Case 3. Ossifying fibroma, oval in configuration and presenting a few scanty striae.

Occasionally the displacement amounted to several centimeters. In 2 cases a loss of the lamina dura was encountered, the roots appearing to lie directly within tumor substance. In several instances partial destruction of tooth roots was also seen. One case showed eradication of the nutrient canal, and in another the canal was displaced slightly inferiorly.

In the 6 tumors of maxillary origin, the nares were found to be affected 5 times, the adjacent orbit and antrum each 4 times, and the anterior ethmoid cells twice. The effect was one of direct encroachment, with a narrow band of bone destruction going on before the advancing tumor. Changes about the teeth were less discernible than in the mandible. In 2 cases teeth were displaced and in one denuded tooth roots were encountered.

The temporal tumor replaced the mastoid process, destroyed the middle ear area, and entered the posterior cranial fossa laterally.

*Condition of Cortex:* All mandibular tumors showed at least moderate expan-

sion, with a thin bony margin continuous with the cortex. Occasionally this expansion was extreme. This thinning and expansion, producing an egg-shell appearance, constituted one of the more striking and constant findings in the smaller ossifying fibromas. In the larger tumors one wonders whether the old cortex of the jaw continues to be expanded and narrowed or whether—and this seemed more likely to us—a new thin bony periphery is constantly reformed around the growing tumor from the periosteal elements believed to be present. In 2 lower jaw tumors small areas of cortical destruction were suspected.

All maxillary tumors showed a thin bony capsule. Only one instance of cortical thinning seemed to be present. In 4 there was destruction of the thin bone forming the antral walls; in one no perceptible cortical effect was present.

**Boundary:** The boundary of every lesion in this series was found to be distinct. It was always possible to separate the tumor from the normal surrounding bone. In 9 instances wide sections of the border took on the appearance of an egg-shell margin. This, however, did not necessarily make up the entire periphery. In the maxillary tumors, for instance, the egg-shell appearance was seen in that part of the tumor not lying adjacent to the bone. Also in the mandibular growths the segments in contact with normal medullary bone usually had no such margin.

**Internal Pattern:** There seemed to be a slight difference in the internal structure or architecture of ossifying fibroma depending on whether it occurred as a mandibular or maxillary process. In 3 of the lower jaw tumors a few fine linear strands of calcification could be identified. These tended to have a radial configuration and 2 showed, in addition, a few ill defined centrally placed areas of calcification. In several others a few tiny scattered areas of poorly defined calcification without any discernible arrangement were seen. In one case there was no internal structure, the process being purely osteolytic.



Fig. 4. Case 4. Ossifying fibroma (enlarged) of temporal bone, replacing mastoid process and extending into middle ear and base of skull.

Three of the maxillary tumors displayed no internal structure or pattern. Of the remainder, one showed fine, delicate radiating strands and two presented only a few scattered calcific flecks.

The ossifying fibroma in the temporal bone showed a few incomplete septa and diffuse structureless areas of increased density (Fig. 4).

It is evident from the foregoing that we have found ossifying fibroma to be a unilocular process. Septa, if present at all, were incomplete and indistinct, never forming true sacculations.

**Multiplicity:** While full skeletal studies were not done in this series, there was no clinical evidence of other tumors, nor was there any radiographic evidence of multiple involvement of bones of the skull.

**Pathologic Fracture:** In 3 cases pathologic fractures were recorded, but in each instance they were small and cortical. There was no complete fracture with displacement of large fragments of the mandible.

ble. The presence of pathologic fracture seemed to be a minor feature and of little significance as relating to x-ray diagnosis or in bringing the patient to the doctor.

*Growth Changes:* The changes that occur in a tumor over a period of time may provide one with the key to diagnosis, because growth effects occasionally produce the most characteristic radiographic features. Because of the slowly progressive character of ossifying fibroma, the roentgenologist is provided at times with a series of films covering a period of months or years. Such interval coverage has been present in several instances in this series.

In Case 7 the tumor was followed for thirteen years. At the time of the initial x-ray study, when the patient was three years of age, the ossifying fibroma measured  $5.5 \times 3.5 \times 3.5$  cm. Thirteen years later the measurement was  $14 \times 12 \times 15$  cm. During this period films were taken at yearly intervals. These displayed a steady tumor growth, which can be summarized as follows: Three years after the initial x-ray examination the tumor was estimated to have increased to six times its original size. Six years after this it was ten times as large. During the first months of observation a single x-ray treatment was given and gold radon seeds totalling 13 millicuries were implanted. There was no other factor which might have influenced growth. We are inclined to consider the amount of radiation as relatively small and having little influence on the course. In Case 11, in which two series of x-ray treatments had been given elsewhere, x-ray studies over a four-year period were available. During this time the lesion showed slow and steady enlargement. A frequent item in the history of these ossifying fibromas has been the discovery of a mass by others than the patient. This also suggests a slow growth.

There was no significant change in the configuration of the tumors throughout the follow-up period.

In 3 cases with radiographic observation for a year or more, there was no doubt that

the ossifying fibromas became denser with the passage of time. This is best demonstrated in the films of Case 7. The productive change in these instances was due to the appearance of circular calcific flecks a few millimeters in diameter. Occasionally these increased by coalescence into masses about a centimeter in diameter. Along with this change, the structureless bed of the tumor seemed to become slightly and diffusely dense. This particular feature is evident in Case 4, which we consider as a relatively old lesion.

We were struck by an unusual growth feature in tumors of the maxilla, best demonstrated in Case 7. Ossifying fibroma of maxillary origin appears to have the ability of growing within the facial bones even though surrounded for the most part by a distinct thin bony margin. It seems to do this by a dissolution of the adjacent bones, the thin intact shell of the tumor being surrounded by a narrow radiolucent zone of bone destruction. We could find no evidence of pronounced pressure effect or displacement in the growing ossifying fibroma as far as the bones of the face were concerned. The ossifying fibroma in the temporal bone showed this type of relation to adjacent bone.

*Summary of Radiographic Features:* Our findings indicate that ossifying fibroma is a lesion occurring generally in the mandible or maxilla, unilocular in type, oval to spherical in configuration, with a distinct boundary, frequently of an egg-shell character. This process in its early phase is predominantly osteolytic, with little or no internal architecture and no periosteal reaction. The tumor originates from the medullary portion of the bone in the lower jaw and either in a similar fashion or subperiosteally in the maxilla. The changes with growth may be fairly characteristic. These are marked by progressive enlargement and an increased productive element brought about in the maxilla by the formation of spherical densities and in the mandible by irregular striae. Ossifying fibroma shows a rather unique



Fig. 5. Case 5. Ossifying fibroma presenting a unilocular, expansile, essentially structureless process.

Fig. 6. Case 6. Ossifying fibroma demonstrable as a unilocular process with egg-shell margin and faint internal structure.

growth change in the maxilla, with dissolution of neighboring bones without pressure displacement.

#### DIFFERENTIAL DIAGNOSIS

Our observations in the 12 cases available for study suggest a dissimilarity in

the radiographic appearance of ossifying fibroma and fibrous dysplasia of bone. It would appear that the fundamental difference is that the former is a unilocular lesion while the latter, when it involves the mandible, is multilocular with distinct and complete septation. We have seen

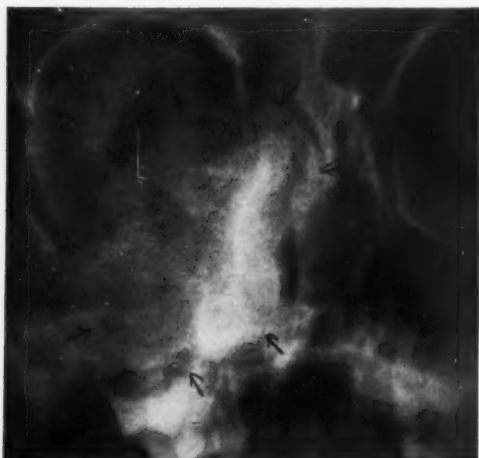


Fig. 7, A. Case 7. Ossifying fibroma of maxilla with fine distinct margin and no internal structure. See also Fig. 7, B and C.

no case of maxillary fibrous dysplasia as a cyst-like process. The type of maxillary involvement we associate with fibrous dysplasia is one in which there is hyperostotic change of the order recently described by Pugh (10). In our cases of fibrous dysplasia in which the mandible was involved along with other bones, the process in the jaw was fusiform or elongated in contrast to ossifying fibroma, which is consistently oval to spherical. In no instance of fibrous dysplasia have we noted the rather unique growth reaction of maxillary ossifying fibroma, namely a tendency for the normal surrounding bones of the facial region to melt away before the expanding bony capsule of the tumor. In those ossifying fibromas that have been followed over a period of years the formation of calcific areas with their later coalescence serves further to distinguish this tumor from fibrous dysplasia. In none of the cases of ossifying fibroma were there skin changes nor were endocrine disturbances mentioned. We have no instance of ossifying fibroma which has become malignant nor have we noted such an occurrence in the literature. Two cases of fibrous dysplasia seen in this hospital (1) have been associated with malignant tumor formation.

Adamantinoma is basically different from ossifying fibroma in that it is a multilocular process. We have recently reviewed about thirty adamantinomas radiographically and consider that this is the essential distinguishing feature, although there are others. Osteitis fibrosa cystica in our experience tends to be multilocular when it involves the mandible. If it is seen as a unilocular area, the absence of a thin egg-shell periphery, absence of calcific deposits or any productive change, and evidence of generalized bone alteration, loss of lamina dura, and altered blood chemistry are specific differential items. The so-called "brown" tumor occasionally seen with osteitis fibrosa cystica is differentiated by its lack of any distinct periphery, absence of cortical expansion, and evidences mentioned above indicating a disease of generalized character.

Our cases of lipoid histiocytosis involving the mandible show no similarity in roentgen appearance to ossifying fibroma: they are multilocular, show cortical destruction, and are elongated, with no egg-shell margin, no calcific flecks, and no distinct boundary.

Central "myxoma," cancer metastases, and osteogenic sarcoma also appear dissimilar radiographically to ossifying fibroma. They show cortical destruction, no septation and no distinct periphery, no cortical expansion, and no egg-shell margin. We have recently reviewed about forty cases of osteogenic sarcoma of the mandible and feel that, in general, this tumor presents the same features in the mandible as permit its identification in the long bones.

Radicular cyst, even though generally a monocystic process, should cause no difficulty in differential diagnosis. This cyst rarely attains the size of ossifying fibroma. It shows neither the free thin egg-shell margin nor productive changes with passage of time. The essential point is the presence of a diseased tooth in intimate association with the cystic area. This relationship is manifested by interruption

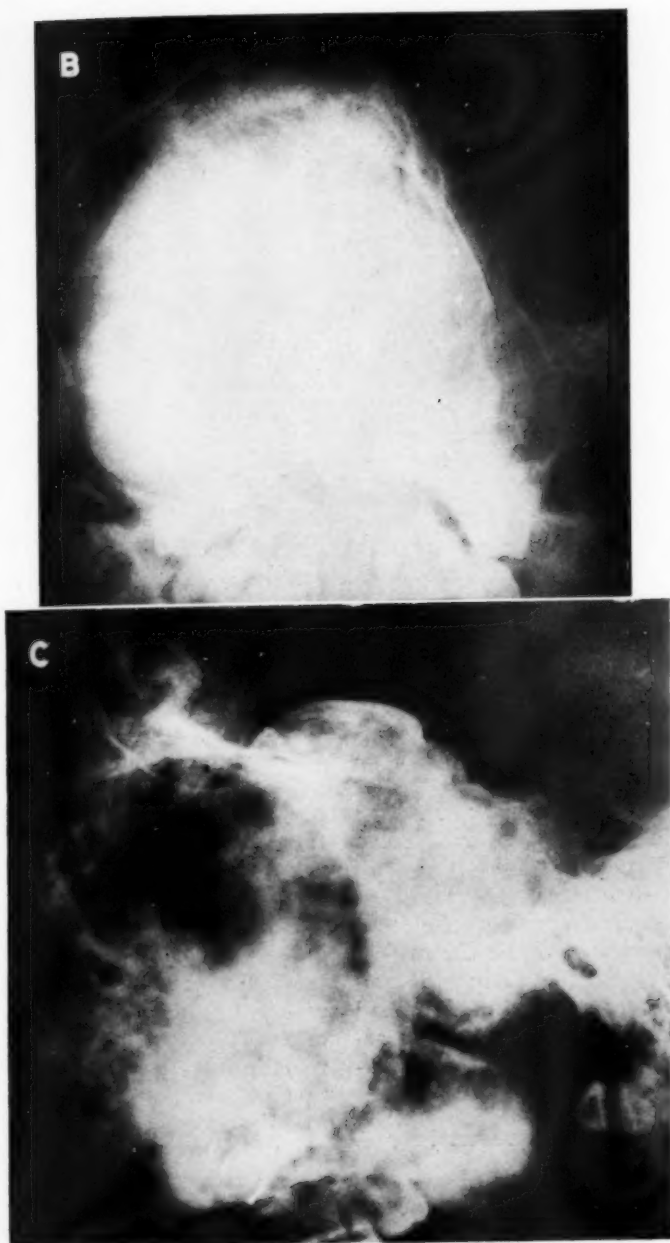


Fig. 7. B and C. Case 7. B. Appearance four years after Fig. 7. A. C. Extent and character of tumor as seen in lateral view thirteen years after initial roentgen study.

of the lamina dura. There is no significant cortical expansion. This cystic lesion usually occurs in adults. Rarely it

may develop after the offending tooth has been removed. The regular, fine dense wall often seen completely sur-

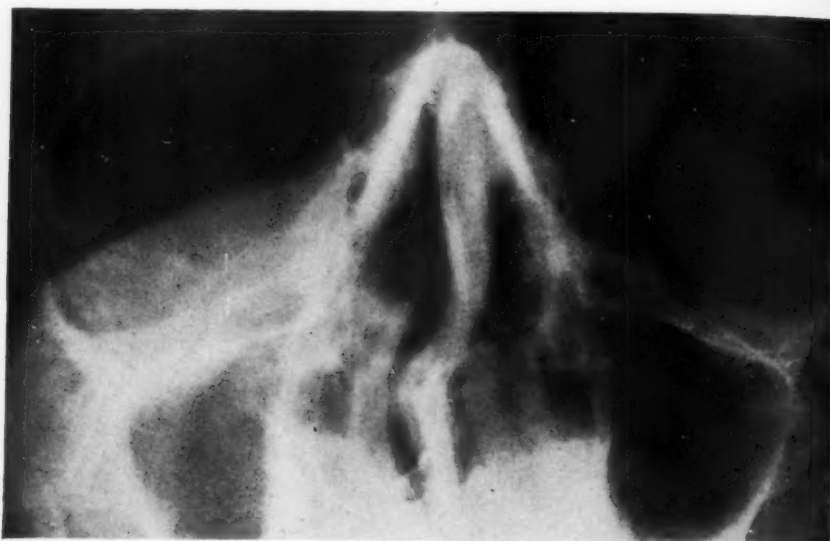


Fig. 8. Case 8. Ossifying fibroma of maxillary origin showing fine capsule and no internal structure.

rounding the radicular cyst then assumes greater importance in differential diagnosis.

Dentigerous cyst is differentiated from ossifying fibroma particularly by the presence of a malformed tooth, usually molar or canine, within the cyst. In those cases where a tooth may not be visualized, other reliable differential points are the absence of a free egg-shell margin, the absence of significant cortical expansion, the continuity of the cyst wall across the medullary area of the jaw, the absence of productive change over a period of time, and the fact that these cysts are usually associated with the eruption of the permanent teeth.

#### CLINICAL OBSERVATIONS; TREATMENT AND RESULTS

Our 12 patients averaged seventeen years of age, the youngest being three and the oldest fifty. Except for the 3 adult cases, the age range was between three and nineteen years.

The chief complaint was the presence of a mass in the facial region. Occasionally there were accompanying symptoms from the growth effects of the tumor, such

as nasal obstruction in 3 patients, proptosis in 2, tearing of the eye in 1, and deafness in 1. Absence of pain associated with the tumor, in spite of the large size that it attained in some instances, is a noteworthy feature. There was an accompanying lack of tenderness. Some of the lesions attained a large size before the patient sought treatment or even became aware of the mass.

Thorough curettage of the tumor area appeared to be a dependable method for treatment of ossifying fibroma. Surgeons reported no difficulty in establishing a line of cleavage. In a few of the larger tumors resection of the part was required. Extensive resection of the maxilla was done in 3 instances and of the mandible in 2 cases.

One patient died twenty-four hours after an attempt to remove a large tumor (Case 7). Two patients have some functional disability after mandibular resections. In 5 of the maxillary and 2 of the mandibular cases the results appear to be excellent. One patient with a mandibular lesion has a rather prominent bony thickening at the operative site but is asymptomatic, with no radiographic signs of recurrence.

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One patient received five x-ray treatments of 300 r each, the details of which are not known to us. This treatment was given before excision was done and seemed to produce no immediate regression of the tumor. In another case, 2,000 r in air were given for a recurrence, and five years after this there was no evidence of disease. In another, relatively heavy x-ray treatment—a total of 3,950 r in two courses—was given elsewhere without discernible effect on the size of the tumor up to the time of surgery three years later. While there was no regression, it may be that there was an arrest of growth, and it seemed to us that there was excessive calcification in this tumor for its size. Case 7 received a single x-ray treatment but the dosage is not known. At least it did not leave permanent skin changes. This was followed by the implantation of 13 mc. of radon through an antrotomy opening. Although there was a close and prolonged follow-up in this case, no beneficial effect of this treatment was noted.

There appeared to be 2 recurrences in the maxilla and 1 in the mandible. In one of these the removal of a benign tumor from the same site ten years previously suggests that the later tumor was probably a recurrence (Case 10). In Case 11 it is possible that the initial operation represented an incomplete removal. Finally, Case 1 has shown recalcification of an area of radiolucence thought to represent a recurrence.

In only 2 cases were complete blood chemistry studies done. The findings were normal.

#### CASE REPORTS

CASE 1: A boy, age 4 1/2, was found to have a swelling of the right lower jaw. A firm, elastic, slightly tender swelling, 4 × 5 cm., was attached to the mandible at the angle. Roentgen studies showed an expanding osteolytic lesion with a sharp, calcified, egg-shell capsule involving the mandible in the vicinity of the angle (Figs. 1A, 1B). The tumor was excised. A year and a half later, a recurrence (Fig. 1C) was given deep x-ray therapy, 2,000 r measured in air. A subsequent five-year follow-up showed no evidence of disease, but there was residual thickening of the mandible (Fig. 1D).

CASE 2: A 14-year-old girl discovered a lump, 2 cm. in diameter, at the angle of the right jaw. This swelling remained painless and non-tender but gradually increased in size and five months later was incised. The patient was referred to Memorial Hospital six months after this, with a markedly swollen jaw. The tumor measured approximately 10 × 10 cm. Examination of the mouth showed displacement of the teeth on the affected side. Roentgen studies depicted an expanding osteolytic lesion involving most of the right side of the mandible (Fig. 2). The tumor was well margined by egg-shell-thin calcification. The right side of the mandible was resected. A ten-year follow-up has revealed no evidence of recurrent disease.

CASE 3: While incising an abscess in the right upper incisor region of a 50-year-old male, a dentist discovered a swelling of the left side of the mandible. The swelling was non-tender and had never been painful. One month later the patient was seen at Memorial Hospital. Many of the teeth were missing and the remainder were in poor repair. The left lower alveolar ridge was 2 cm. in width and the swelling was 5 cm. in length. Roentgen studies showed an expanding osteolytic lesion (Fig. 3). Incomplete septa were seen extending centrally from the cortex. An intraoral view showed an egg-shell capsule. The tumor was excised and there was no evidence of recurrence seven and one-half years later.

CASE 4 (from Jefferson Hospital, Department of Radiology, Dr. Paul C. Swenson, Director): A boy of 15 years was referred to the outpatient department of Jefferson Hospital by the school doctor because of deafness of the right ear. His mother had noticed a prominence behind the ear and thought it had become somewhat larger over a period of years. The mass was stony hard and, to the mother's knowledge, had always been so. During eight months' observation there was no change in size of the mass. A biopsy was then done, and at operation a thin outer shell was broken and serous fluid was obtained. The wall of the mass was curetted. In x-ray appearance this case differs a little from the other ossifying fibromas in this group because of the somewhat greater amount of slightly dense structureless matrix (Fig. 4). As this is a current case, there is as yet no follow-up.

CASE 5: A boy of 8 years had a painless swelling of the left jaw five months before admission to Memorial Hospital. A hard, non-tender mass, 2 cm. in diameter, was palpable in the region of the angle of the mandible. Roentgen studies showed an expanding osteolytic lesion (Fig. 5), and in the anteroposterior projection a well defined egg-shell capsule was demonstrable. The tumor was excised. Four and one-half years after operation there was no evidence of recurrence.

CASE 6: Three months before admission a 12-year-old boy was found to have a swelling of the left side of the lower jaw, which was attributed to



Fig. 9. Case 9. Laminagraph through maxillary ossifying fibroma.

the teeth. One tooth was extracted and found to be normal. On admission to Memorial Hospital the ramus and body of the mandible were found to be replaced by a bony hard swelling, ovoid in form, with some disturbance of the dental arch. Roentgen studies (Fig. 6) showed an expansile process destroying most of the bone, and having an egg-shell capsule. The left half of the mandible was resected. Three years following treatment there is no evidence of disease.

**CASE 7:** A 3-year-old boy was seen because of swelling of the right cheek, tearing, and right exophthalmos of six weeks' duration. The right naris was blocked. Roentgen studies showed a discrete tumor of water density, which had destroyed the infraorbital plate and occupied the right antrum (Fig. 7, A). An aspiration biopsy was done, and the report was "low-grade osteogenic sarcoma." A month after the child was first seen a single x-ray treatment (dosage not known) was given. Three months later an antrotomy was done and 4 gold radon seeds, totalling 13 mc., were inserted into the tumor. Two years after the first roentgen study the tumor showed a more distinct egg-shell membrane. Two years later it had enlarged to six times its previous size and showed amorphous calcification (Fig. 7, B). The patient was followed for thirteen years and during this time the tumor enlarged progressively, keeping a well defined egg-shell capsule but "melting away" the bony structures in its path as it grew (Fig. 7, C). A review of the original biopsy was made twelve years after the first hospital admission and the diagnosis of osteogenic sarcoma was changed to ossifying fibroma. Because of the patient's grotesque features, an attempt was made to remove a part of the tumor, but death occurred twenty-four hours postoperatively.

**CASE 8:** A boy of 16 years first noticed a non-tender painless swelling of the right cheek six months before admission. Three months prior to admission his physician had taken a biopsy, after which five deep x-ray treatments of 300 r each were given without visible effect. The tumor encroached on the floor and lateral wall of the nose and measured  $6 \times 5$  cm. Roentgen studies (Fig. 8) showed a well defined tumor with an egg-shell capsule, extending from the anterior wall of the antrum. The tumor was excised. At a follow-up examination two years and ten months after treatment



Fig. 10. Case 10. Ossifying fibroma of the left antrum, with distinct periphery.

the patient was free of disease. Two years later two buccogingival fistulae appeared at the site of the previous operation.

**CASE 9:** A 9-year-old boy was found to have a left paranasal swelling of one month's duration, which was painless and non-tender. Examination revealed a 2-cm. bony-hard tumor fixed to the underlying structures. The overlying skin was freely movable. The nasal cavity was blocked superiorly. Roentgen studies showed a sharply outlined oval mass in the left paranasal area, associated with slight bone destruction of the left nasal process (Fig. 9). The tumor encroached on the anterior ethmoid cells, antrum, orbit, and nasal cavity. The mass was removed through the gingival-buccal gutter. A two year follow-up disclosed no evidence of disease.

**CASE 10:** A 37-year-old female had her attention called to a swelling of the left alveolar ridge by her dentist one month before admission. She had not been aware of the swelling, but ten years previously a benign tumor had been removed from the same site. Roentgen studies showed a rounded, discretely outlined tumor destroying the lateral wall of the antrum (Fig. 10). The capsule suggested an egg-shell membrane. The mass was excised. A two and one-half year follow-up showed no evidence of disease.

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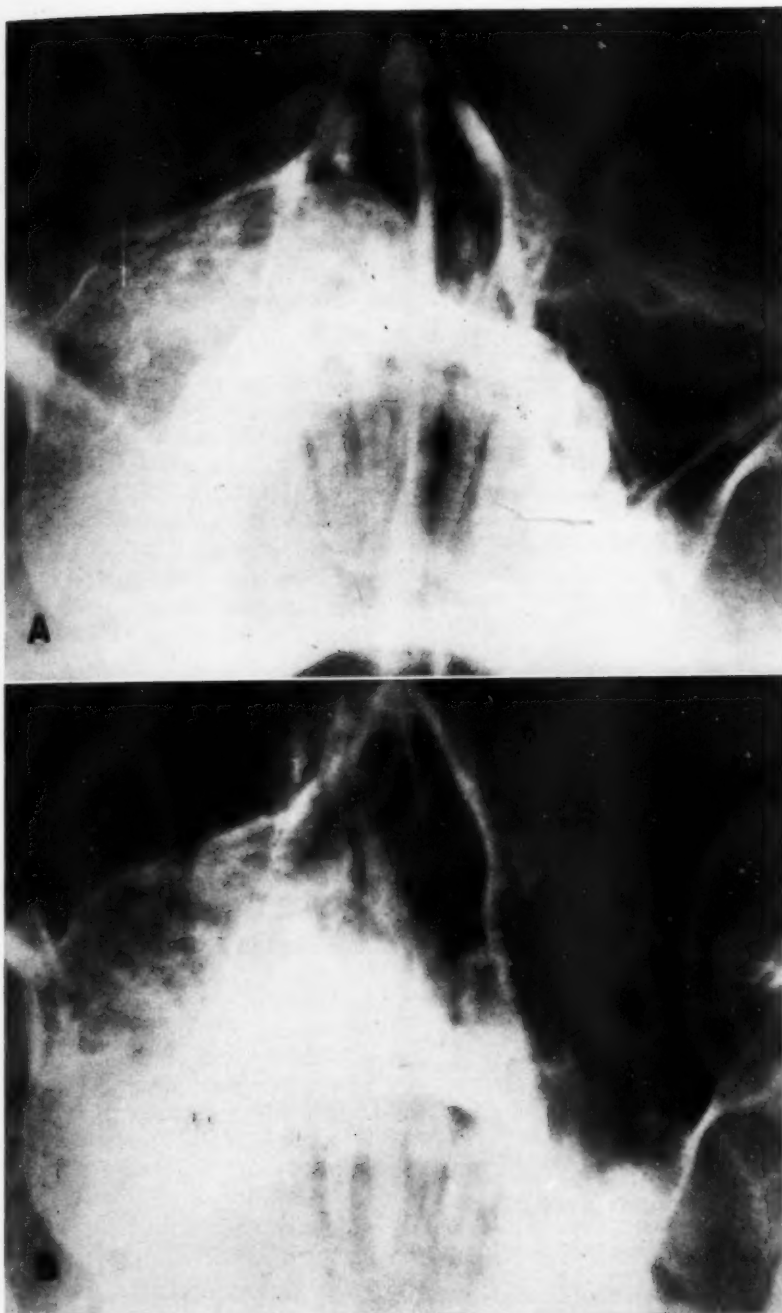


Fig. 11. Case 11. A. Six years after initial symptoms this ossifying fibroma shows increased density. This represents an older tumor. B. Four years later only slight increase in size and greatly increased calcification are seen. These findings may be due to previous x-ray treatment.



Fig. 12. Case 12. Ossifying fibroma of maxilla.

CASE 11: A male, aged 19, presented himself with a swelling of the right cheek which was first discovered during a routine school examination ten years previously. At that time the tumor had been incised through an antrotomy. Three years later tearing of the right eye and right nasal obstruction developed. Three years after this the condition was diagnosed as fibrosarcoma and roentgen therapy was instituted, two series of deep x-ray treatments being given through a  $5.5 \times 5.5$  cm. portal. In the first series the total dose was 1,700 r measured in air and at the second 2,250 r. No decrease was noted in the size of the tumor. Three and one-half years later the patient began to experience pain in the forehead. It was thought that the slight exophthalmos present on the right was becoming greater, suggesting growth of the tumor. The first roentgenogram available, taken immediately before x-ray treatment, showed a discrete rounded tumor with amorphous calcification and an egg-shell capsule, occupying the right antrum and encroaching on the medial and anterior walls and infraorbital plate (Fig. 11, A). Interval studies showed gradual enlargement (Fig. 11, B). The tumor was excised and there had been no evidence of recurrence after three and one-half years.

CASE 12: A 24-year-old female first noticed slight enlargement in the right upper molar region six months prior to admission to Memorial Hospital. Several well encapsulated pieces of tissue, varying from 0.5 to 1.5 cm. in size, had been removed elsewhere and diagnosed "central fibroma." Nasal congestion continued and enlargement of the right gingival margin progressed. Examination revealed a bony bulging tumor about 4 cm. in diameter arising from the lateral surface of the alveolar process

and the maxilla. X-rays showed an oval mass in the right antral area having a distinct egg-shell periphery and containing fine scattered amorphous areas of calcification. The roof of the antrum was partially destroyed, and the tumor bulged into the right orbital floor. It had also destroyed the adjacent alveolar and lateral antral walls. Roentgen diagnosis of ossifying fibroma was made (Fig. 12). A firm, lobulated, well encapsulated tumor, measuring  $6.5 \times 5 \times 3$  cm., was removed at operation.

#### SUMMARY

Twelve cases of ossifying fibroma of bone have been studied, primarily from the point of view of roentgen diagnosis. Six involved the maxilla, 5 the mandible, and 1 the temporal bone. The clinical and radiographic features are believed to be sufficiently distinct to warrant acceptance of ossifying fibroma as a separate entity.

The roentgen findings are usually suggestive of the diagnosis and in certain instances may be characteristic. The picture is that of a unilocular lesion, in most cases limited to the maxilla or mandible oval to spherical in shape, with a distinct boundary, usually of an egg-shell character. In the lower jaw the origin is from the medullary portion of the bone; in the upper jaw the tumor arises in similar fashion or subperiosteally. In its early phase the process is predominantly osteolytic, with little or no internal architecture and no periosteal reaction. Enlargement is progressive, with an increased productive element brought about in the maxilla by the formation of spherical densities and in the mandible by irregular striae. A rather unique growth change in the maxilla is the dissolution of adjacent bone without pressure displacement.

A brief discussion of clinical, pathological and treatment aspects, particularly as they bear on roentgen diagnosis, has been included.

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#### BIBLIOGRAPHY

1. COLEV, B. L., AND STEWART, F. W.: Bone Sarcoma in Polyostotic Fibrous Dysplasia. *Ann. Surg.* 121: 872-881, 1945.
2. EDEN, K. C.: Benign Fibro-osseous Tumours of the Skull and Facial Bones. *Brit. J. Surg.* 27: 323-350, 1939.

3. FOOTE, F. W.: Personal communication.
4. GESCHICKTER, C. F.: Tumors of Jaws. *Am. J. Cancer* **24**: 90-126, 1935.
5. HEATH: Injuries and Diseases of the Jaws. London, J. and A. Churchill, 1894.
6. LICHTENSTEIN, L.: Polyostotic Fibrous Dysplasia. *Arch. Surg.* **36**: 874-898, 1938.
7. MALLORY, T. B.: Medical Progress: Pathology, Diseases of Bone. *New England J. Med.* **227**: 955-960, 1942.
8. MONTGOMERY, A. H.: Ossifying Fibromas of the Jaw. *Arch. Surg.* **15**: 30-44, 1927.
9. PHEMISTER, D. B., AND GRIMSON, K. S.: Fibrous Osteoma of the Jaws. *Ann. Surg.* **105**: 564-583, 1937.
10. PUGH, D. G.: Fibrous Dysplasia of Skull; Probable Explanation for Leontiasis Ossea. *Radiology* **44**: 548-555, 1945.
11. SCHLUMBERGER, H. G.: Fibrous Dysplasia (Ossifying Fibroma) of the Maxilla and Mandible. *Am. J. Orthodontics (Oral Surg. Sect.)* **32**: 579-587, 1946.
12. SCUDDER, C. B.: Tumors of the Jaw. Philadelphia, W. B. Saunders Co., 1912, p. 140.

## SUMARIO

## Aspecto Roentgenológico del Osteofibroma Osificante

Doce casos de osteofibroma osificante fueron estudiados, primordialmente desde el punto de vista del diagnóstico roentgenológico. Seis afectaban el maxilar, 5 la mandíbula inferior y 1 el temporal. Las características clínicas y radiográficas parecen ser suficientes para justificar la aceptación del fibroma osificante como entidad patológica independiente.

Los hallazgos roentgenológicos suelen sugerir el diagnóstico y en ciertos casos pueden ser típicos. El cuadro es el de una lesión unilocular, en la mayor parte de los casos limitada a una de las mandíbulas, de forma oval a esférica, con límites bien definidos, y por lo general de la naturaleza del cascarón de huevo. En la mandíbula inferior el punto de origen radica en la porción medular del hueso; en la superior es semejante o radica en el subperiostio.

En su fase incipiente el proceso es de predominio osteolítico, con muy poca o ninguna arquitectura interna y sin reacción perióstica. La hipertrofia es progresiva, con aumento del elemento productivo, ocasionado en el maxilar por la formación de espesamientos esféricos y en la mandíbula inferior de estrías irregulares. Una alteración algo única en el maxilar consiste en la disolución del hueso adyacente sin desplazamiento por presión.

En esta serie 3 de los enfermos eran adultos; la edad de los demás variaba de tres a 19 años. El síntoma principal consistía en la presencia de una tumefacción, sin dolor ni hiperestesia. El raspado completo de la zona afectada resultó ser un método terapéutico fidedigno, aunque en algunos casos el tamaño era tal que impuso una resección extensa del hueso.



## Importance of Intra-Alveolar Pressure in the Diagnosis of Pulmonary Diseases<sup>1</sup>

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THE STRUCTURE of the lungs and hilum is determined, in the main, by the pulmonary vessels. The roentgen appearance of the lungs is, therefore, to a great extent dependent upon the degree of filling of these vessels. With an increase in filling, the lung pattern becomes more prominent; with a decrease, it is less pronounced. Even the small variations in the intra-alveolar pressure that occur during normal respiration affect the filling of the vessels. During inspiration, when the pressure falls, the filling of the vessels increases, and during expiration, when the pressure rises, it decreases.

The influence of the intra-alveolar pressure on the filling of the vessels and on the structure of the lungs is also proved by the observations made in Valsalva's and Müller's experiments. In Valsalva's experiment the intra-alveolar pressure increases and the filling of the vessels in the lungs decreases. In roentgenograms made during this experiment we see a considerably decreased vascular structure in the hilum and the lungs. In Müller's experiment the intra-alveolar pressure decreases and vascular filling increases. In roentgenograms made in the course of this experiment the vascular shadows appear more pronounced than normal.

Evidence that an increase in the intra-alveolar pressure causes the degree of filling of the vessels to decrease is afforded by observations made under conditions of bronchostenotic emphysema, in which there is an increased intra-alveolar pressure with anemia of the lungs. That decreased intra-alveolar pressure causes increased filling is shown by observations made in obstructive atelectasis, in which

there is a decreased intra-alveolar pressure and hyperemia of the affected portion of the lung.

The varying intra-alveolar pressures during respiration also influence the blood flow to the auricles and to some extent the flow of blood from the ventricles. During inspiration, when the pressure falls, the blood is sucked through the venae cavae to the thorax, and the right auricle is filled. During expiration the flow to the right auricle decreases or ceases. The blood flow to the left auricle, on the other hand, decreases during inspiration and increases during expiration. The outflow from the ventricles is rendered easier during inspiration and more difficult during expiration. The greater the variations in the intra-alveolar pressure, the more pronounced is its influence on the circulation and, in consequence, on the heart. In Valsalva's experiment, the flow of blood to the right auricle, and the outflow from the right ventricle, is even more difficult, and the volume of the heart can be observed to decrease during this experiment. In Müller's experiment the conditions are reversed.

During the course of the usual roentgen study of the lungs there may be marked variations in the intra-alveolar pressure from one patient to another or from one examination to another in the same patient. Sometimes, when the patient is directed to stop breathing, he will strain a little while closing the glottis. As a result, the intra-alveolar pressure will rise and become positive despite the fact that the film is to be made in the phase of inspiration. On the other hand, he may continue to inspire after closure of the glottis, thus

<sup>1</sup> Presented at the Thirty-second Annual Meeting of the Radiological Society of North America, Chicago, Ill., December 1946. To be published in full in a forthcoming book, *Roentgen Studies of the Lungs and Heart*, University of Minnesota Press.

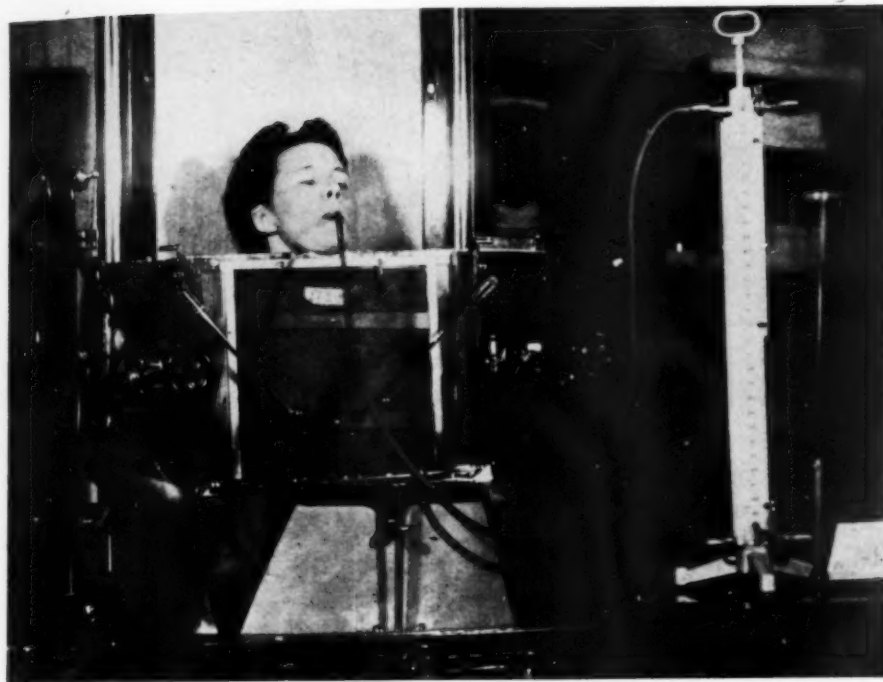


Fig. 1. The manometer in use. Note the manner in which the tube is held in the patient's mouth and the connection with the manometer.

producing a negative intra-alveolar pressure. The films obtained will give a distinctly different appearance under these two conditions although the patient is presumably attempting to follow the same directions.

Laurell has pointed out that it is necessary to produce the same intra-alveolar pressure each time an examination is made if the roentgenograms of the lungs are to be comparable. Therefore, he has suggested the use of an ordinary water manometer for facilitating the production of sharp roentgenograms and for the production of films of the lungs under the same conditions of pressure. The one shank of the manometer is connected by a rubber tube and a mouthpiece with the patient's mouth, and the patient is then instructed to fix the water level at a certain height, which must be the same in the various examinations. If this procedure is correctly done, so that the air in the bronchi is in connection with the

manometer, there will be an oscillatory movement of the surface of the liquid which is synchronous with the pulse rate of the person examined. The oscillations are caused by the heart pulsations, which are transmitted to the trachea. If these oscillations are not discernible, the air in the bronchi must be shut off from the manometer.

In using the water manometer, as suggested by Laurell, at the St. Göran Hospital, (Fig. 1), we employ for the usual roentgen examination a normal pressure of  $-5$  to  $-10$  cm. of water. Immediately before the roentgenogram is made, the patient is told to take a shallow inspiration, and then to suck through the mouthpiece until the level of the liquid falls so that a pressure of  $-5$  to  $-10$  cm. of water is reached. To obtain films at low pressure, the patient is first requested to take a light inspiration, and then to augment the inspiratory movement with closed mouth and nose until the pressure falls to

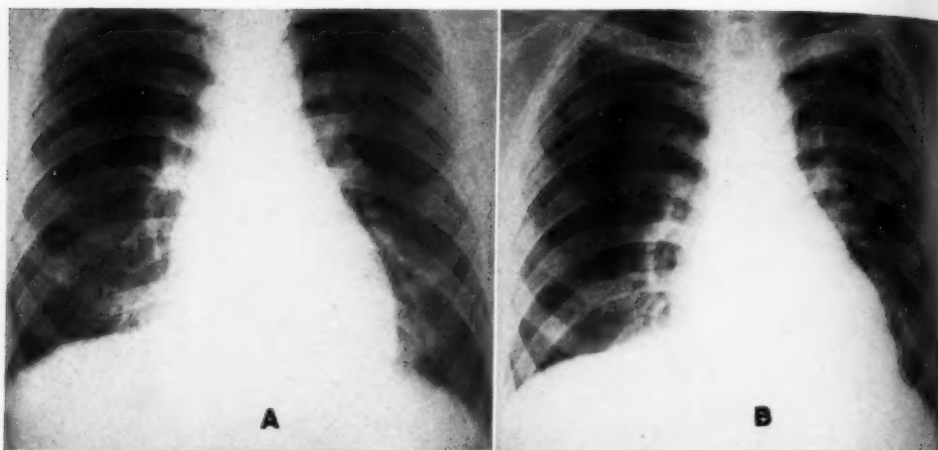


Fig. 2. Mitral stenosis of minor degree. A. Roentgenogram made with normal intra-alveolar pressure. B. Roentgenogram made with high intra-alveolar pressure.

The central branches of the pulmonary artery are shown to be compressed in the high-pressure roentgenogram (B), as compared to their size under normal conditions (A). Note the diminution in the size of the heart in the film made under high pressure (B).

—40 cm. To obtain films when there is a high pressure, the patient is again asked to take a light inspiration. He must then, by blowing with closed mouth, press the water level of the manometer up to a positive pressure of 40 cm. of water. These different pressures are employed in routine examinations. If, on any occasion, another pressure is used, this is indicated separately.

When comparing the roentgenograms taken under normal pressure with those obtained with a low pressure, we find that the latter demonstrate a considerably increased filling of the vessels in the lungs, an increased root shadow, and a larger cardiac volume. On the other hand, in the film made under high pressure, compared with the film taken under normal pressure, there is a considerably reduced filling of the vessels, the vascular structure of the lungs appears to be diminished, and the cardiac volume is decreased. Thus we have been able to demonstrate that the volume of the heart can increase by up to one-third when the pressure is low, and decrease by one-third when a high pressure exists. The size of the heart consequently depends upon the intra-alveolar pressure employed.

By many experiments I have established that in normal cases a positive intra-alveolar pressure of 40 cm. of water, or approximately 30 mm. of mercury, is required to compress the central branches of the pulmonary artery. This pressure corresponds to the intravascular pressure in the pulmonary artery during the experiment. In the production of this high pressure, however, the flow of blood to the right auricle is influenced, and becomes less copious. The decreased supply of blood tends to lower the pressure in the pulmonary artery. However, the high intra-alveolar pressure also affects the pulmonary capillaries, which become compressed. This condition tends to increase the pressure in the pulmonary artery. These two factors, the decreased flow to the right auricle and the compression of the pulmonary capillaries, thus counteract each other in their influence on the blood pressure in the pulmonary artery. Provided the conditions of the experiments are equal when the high pressure is produced, its influence on the flow of blood to the right auricle and on the capillaries may be regarded as constant. Thus, the difference between the effect of the high intra-alveolar pressure in causing a decrease and an increase of

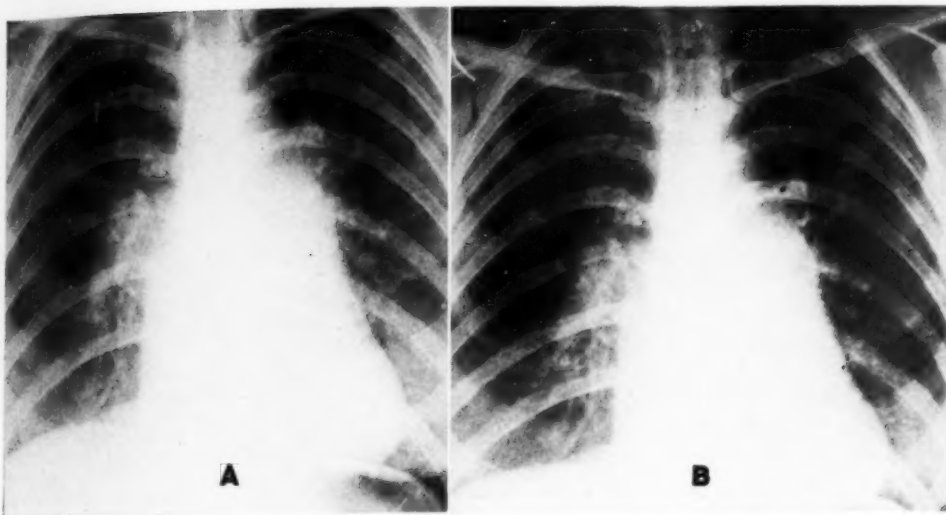


Fig. 3. Severe mitral disease. A. Roentgenogram with normal pressure. B. Roentgenogram with pressure of 80 cm. of water.

There is no change with the increased pressure. The pressure within the pulmonary artery is so high that it is able to resist even a pressure of 80 cm. of water in the alveoli.

pulmonary artery pressure is also constant. Consequently there is a fairly constant relation between the blood pressure in the pulmonary artery and the amount of intra-alveolar pressure required to compress the central branches. This relationship is influenced by the difference between the intra-alveolar pressure and the intratracheal pressure resulting from the elasticity of the lung.

Even though this method of determining the pressure in the pulmonary artery may be a rough one, it is of practical importance. In the examination of 90 cases of mitral disease, for example, I have found in mild cases a normal pressure in the pulmonary artery (Fig. 2). In more severe cases, the pressure is more or less augmented and in very serious cases it may reach a considerable height (Fig. 3). The method is thus of importance for the determination of the degree and the prognosis of mitral valve disease. It may also be valuable for the diagnosis of other diseases causing increased blood pressure in the pulmonary artery.

The fact that we can employ a high pressure to compress the central branches of

the pulmonary artery enables us to demonstrate clearly small lymph nodes in the hilum area on the roentgenogram of the chest (Fig. 4). Large nodes, also, emerge more distinctly and are easier to delimit in films made under high intra-alveolar pressure than in those made with normal pressure. Many a time it may be difficult to decide whether a shadow is caused by a hilar node or a dilated vessel. In these cases, the diagnosis is easily determined if high-pressure roentgenograms are made; the node is not affected by the high pressure, whereas the vessel is compressed. By taking films under both normal and high pressures, we can make a more accurate diagnosis of hilar node enlargement.

In a study of primary tuberculosis with positive tuberculin reaction and positive bacillary findings, hilar node enlargement was demonstrable in 62 per cent of the cases. This study was made on earlier material, and roentgenograms were obtained under normal pressure only. In a later series of cases of erythema nodosum of primary tuberculous origin in which roentgen examinations were made with both normal and high intra-alveolar pres-

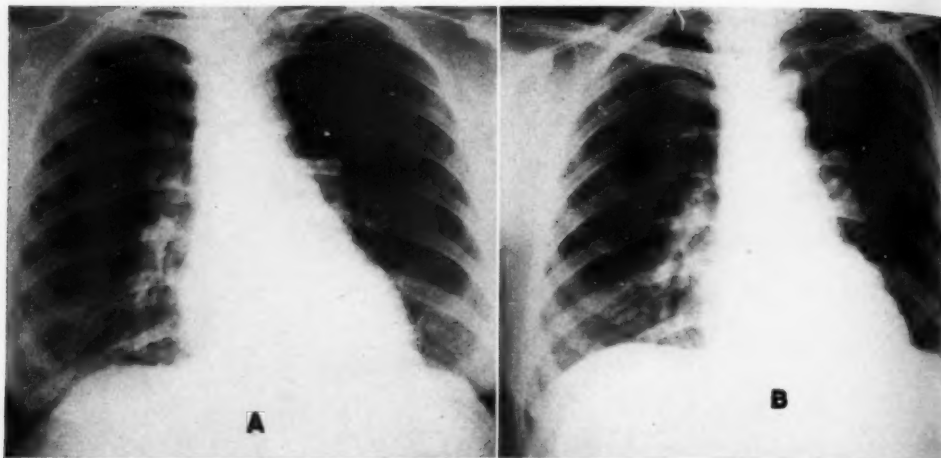


Fig. 4. Hilar node enlargement. A. Roentgenogram with normal pressure. B. Roentgenogram with high pressure.

Note the apparent absence of lymph node enlargement in A while it is clearly shown in the inferior portion of the left hilum in B.

sure, enlargement of the hilar nodes was found in 97 per cent of the cases. The higher figure was probably due altogether to the method of examination.

During inspiration, the contraction of the diaphragm causes an enlargement chiefly of the basal and dorsal parts of the thoracic cavity, while the movements of the wall of the chest induce an expansion especially of the anterior and lateral parts of the lung. Under normal conditions, however, the lobes of the lungs move in relation to each other, just as the entire lung is displaced toward the thoracic wall and the diaphragm. By filling the bronchial tree with opaque medium, we can observe that the whole lung rotates on a frontal axis, so that the lower parts rotate forwards and the upper lobe backwards. Through such displacements, the expansion of the various parts of the lung is balanced so that it is comparatively uniform. Under normal conditions the filling of the vessels thus increases uniformly in the lungs upon inspiration. In the presence of adhesive pleurisy, this movement of the lobes and of the lung and the thoracic wall cannot take place. In such a case the filling of the vessels therefore varies in the different parts of the lung.

Under pathological conditions a hyperemia may develop in the lungs, or parts thereof. Hyperemia can thus result from stasis in the pulmonary circulation or from inflammatory changes in the lungs. It would seem reasonable that high intralveolar pressures might be able to influence such hyperemia, just as it can influence the normal vascular filling of the lungs. At the St. Göran Hospital we have, for this reason, during the last ten years routinely subjected patients showing pathological changes in the lungs to examinations under both normal and high pressures. Approximately 15,000 cases have now been examined by this method.

In lung stasis we have established that the widened vascular shadows and the opacities caused by pulmonary edema decrease considerably with the application of high pressure (Fig. 5). In certain cases the appearance of stasis may be entirely obliterated; in others, it becomes less pronounced than under normal pressure. The pulmonary edema also diminishes considerably, so that the more or less massive opacities present are reduced both with regard to intensity and size.

In cases of acute bronchopneumonia, it has been found that the diffuse and opaque

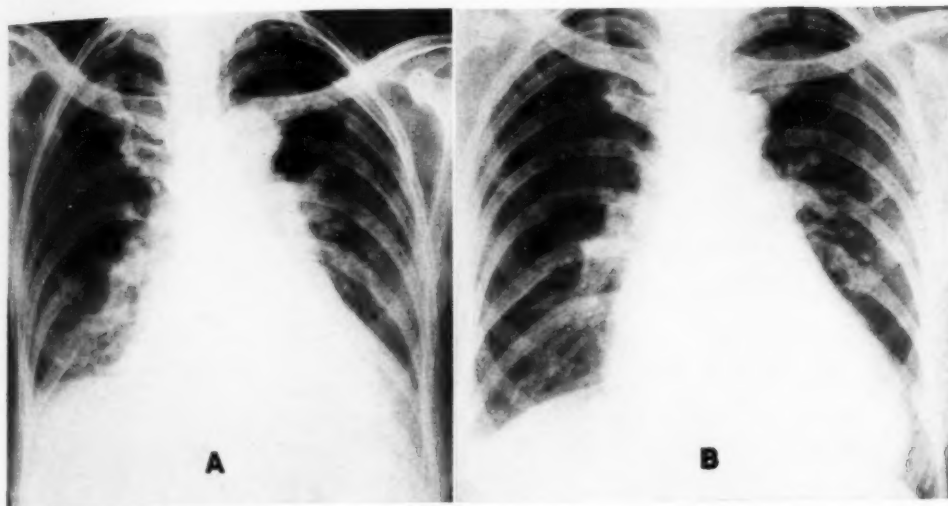


Fig. 5. Pulmonary congestion. A. Roentgenogram with normal pressure. B. Roentgenogram with high pressure.

In the normal pressure roentgenogram (A) there is shown a definite increase of the vascular pattern and numerous minor shadows indicating edema and a small amount of fluid in the pleural cavity. In the high-pressure film the appearance is also normal.

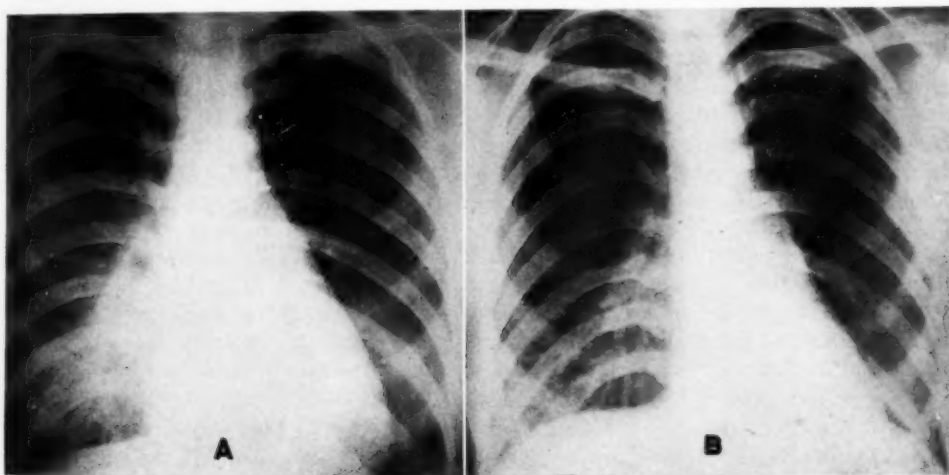


Fig. 6. Acute pneumonia in the right middle lobe. A. Roentgenogram with normal pressure. B. Roentgenogram with high pressure.

With normal pressure (A) the diffuse opacity caused by the pneumonia is clearly shown. Under high pressure (B) the shadow has almost completely disappeared.

network of the parenchyma, which is visible in roentgenograms taken under normal pressure, appears thinner in films made at high pressure, so that the opacity becomes less pronounced and more sharply defined (at the same time becoming more distinct in outline). In many instances

this increase in clarity may be considerable; in others it is less distinct.

In many cases of acute lobar pneumonia, also, a considerable decrease in opacity is observed under high pressure. Under normal pressure, the opacity may be diffuse. With the application of high pres-

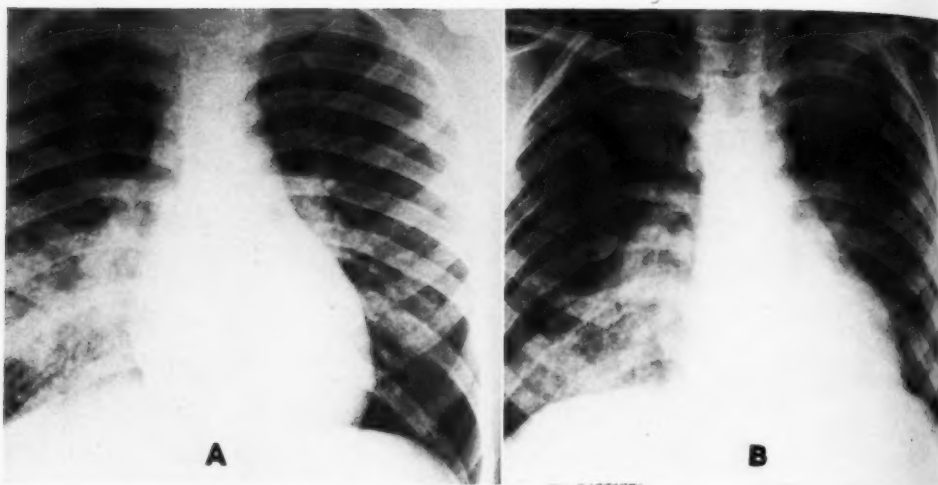


Fig. 7. Acute pneumonia seven days after the onset. A. Roentgenogram with normal pressure. B. Roentgenogram with high pressure. With normal pressure (A), there is a pneumonic opacity in the right lower lobe. With high pressure (B), the opacity is still shown but it is more distinct and more clearly outlined.

sure, there is a decrease in intensity and size (Fig. 6), and the opacity is more distinctly outlined and more sharply defined (Fig. 7). In cases of lobar pneumonia in the later stages, no very great difference is seen in roentgenograms taken under normal and high pressures. This is particularly true of the massive lobar pneumonias. Sometimes, however, we observe even in these cases a decrease in the size of the infiltrated region, while opacities that may exist in the nearby portions of the lung may at the same time be found to be more distinctly marked, and more sharply defined, under high pressure than under normal pressure.

As a rule, no very great difference is noticeable in cases of acute and chronic obstructive atelectasis. In the acute forms, however, we sometimes observe a decrease in volume of the atelectatic region, while the opacity becomes more sharply defined against the surrounding lung parenchyma. In chronic cases no change occurs.

In acute primary tuberculosis, there generally appears no very great difference in the opacity of the parenchyma. However, in certain instances the parenchymal shadow may appear more distinctly out-

lined and defined with high pressure than under normal pressure, though the difference is not so pronounced as in the acute bronchopneumonias. The regional node shadows in the hilum, however, are more clearly displayed after high pressure, and more sharply delimited than under normal pressure. In acute exudative tuberculosis, there is, as a rule, no very great difference, but the change is sometimes a little more clearly defined after high pressure than under normal pressure. In chronic tuberculosis, I have been unable to observe any differences in the films.

Comparing roentgenograms made under conditions of normal intra-alveolar pressure with those taken under high pressure, we thus find that a definite difference is evident in pulmonary stasis, pulmonary edema and in acute pneumonic changes. In chronic inflammatory conditions and tuberculosis, there are no changes; or, in any case, less striking changes than in acute conditions. By the introduction of a routine involving the taking of films under both normal and high pressure, a more exact and safer diagnosis can thus be obtained than if the examination were carried out under normal pressure only.

This investigation also shows that in the ordinary procedure of chest roentgenography it is necessary to make roentgenograms under the same conditions of intra-alveolar pressure. For example, it is possible for the patient when he stops breathing in the ordinary manner to strain a little when closing the glottis. In that case there is produced a positive intra-alveolar pressure. On the other hand, he may continue the inspiratory movements after closing the glottis, in which case there is then produced a negative intra-alveolar pressure. The appearance of the normal lung may be different in these situations, with the result that misinterpretations may occur.

#### SUMMARY

1. The degree of filling of the pulmonary vessels, of the capillaries, and of the chambers of the heart is profoundly affected by the intra-alveolar pressure.

2. In order to obtain roentgenograms at various intervals in which the vascular structures maintain the same size, it is necessary to make films of the chest under standard conditions of intra-alveolar pressure. This is done by using a water manometer with a tube in the patient's mouth. Pressures of  $-15$  cm. of water up to  $+80$  cm. of water can be obtained.

3. With the use of such a manometer,

films may be made at normal levels and at high pressures and then compared. A rough estimate of the pressure in the pulmonary arteries can be obtained in this way by determining the point at which the arteries in the hilum are diminished in size.

4. Roentgenograms made during positive intra-alveolar pressure may reveal lymph nodes in the hilum not otherwise demonstrable, the diminished size of the vessels making the nodes more easily visible.

5. In moderate degrees of congestion and in pulmonary edema films made under high-pressure conditions will result in sharp diminution or disappearance of the shadows.

6. In acute bronchopneumonia in an early stage, faint shadows of the process may disappear when pressure in the alveoli is elevated.

7. In acute lobar pneumonia of massive type, and in acute and chronic atelectasis, in chronic pneumonia, and in most cases of tuberculosis, changes are minor or absent altogether when the pressure is changed.

8. In routine x-ray examinations of the lungs, standard intra-alveolar pressure should be assured, so that accurate comparisons can be made.

St. Göran's Hospital,  
Stockholm, Sweden

#### SUMARIO

#### La Importancia de la Presión Intraalveolar en el Diagnóstico de las Afecciones Pulmonares

El henchimiento en mayor o menor grado de los vasos y los capilares pulmonares y de las cavidades del corazón se ve profundamente afectado por la presión intraalveolar. A fin de obtener radiografías a varios plazos en las cuales los órganos vasculares retengan el mismo tamaño, es necesario, por lo tanto, hacer películas torácicas a la misma presión intraalveolar. Se logra esto utilizando un hidromanómetro y colocando un tubo en la boca del enfermo, lo cual permite obtener presiones desde  $-15$  cm. hasta  $+80$  cm. de agua.

Empleando dicho manómetro, pueden obtenerse radiografías a cifras normales y a presión alta, comparándolas después. Determinado el punto en el cual las arterias del hilio disminuyen de tamaño, puede hacerse de este modo un cálculo aproximado de la presión en las arterias pulmonares.

Las radiografías obtenidas con una presión intraalveolar positiva pueden revelar en el hilio ganglios linfáticos inobservables de otro modo, por ponerlos más de relieve el atenuado tamaño de los vasos.

Si hay congestión moderada o edema

pulmonar, las películas obtenidas a una presión alta mostrarán disminución aguda o desaparición de las sombras.

En la bronconeumonía aguda en su período incipiente, pueden desaparecer las sombras débiles al elevarse la presión.

En la neumonía lobular aguda de tipo masivo y en la atelectasia aguda y crónica,

la neumonía crónica y la mayor parte de los casos de tuberculosis, las alteraciones son banales o faltan por completo al variar la presión.

En los exámenes corrientes de los pulmones con los rayos X, hay que mantener una presión intraalveolar constante a fin de poder hacer comparaciones exactas.



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# Bone and Joint Lesions in Leprosy

## A Radiologic Study<sup>1</sup>

PROF. GONZALO ESGUERRA-GÓMEZ and DR. EMILIO ACOSTA

Bogotá, Colombia

IN THE FEW PATIENTS with leprosy presenting themselves at the x-ray laboratories in Bogotá, the majority without a clinical diagnosis of the disease, an opportunity was afforded to observe in the bones of the hands and feet trophic lesions sufficiently characteristic to make possible a radiologic diagnosis. In view of these observations, it was decided to undertake a more complete study of the bone changes in leprosy. This was the more easily accomplished since in Colombia lepers are isolated in leprosariums, a practice due rather to the traditional fear of the disease than to any actual danger of contagion.

My distinguished pupil and collaborator, Dr. Emilio Acosta, served for two years as head of the X-ray Department at the lazaretto in Agua de Dios, where he not only attended those patients requiring x-ray service but also obtained films of the hands and feet which have contributed greatly to our investigation.

Between 1932 and 1940 there were published three radiological reports covering 231 cases of leprosy (Murdock and Hutter; Karaseff; Oberdoerffer and Collier). Karaseff and Murdock and Hutter were of the opinion that the bone lesions of leprosy are located exclusively in the hands and feet. Dr. Acosta, after studying over 1,000 lepers confirmed this view, adding: "Of the organs examined, only the larynx showed some suspicious changes. There occur a variety of lesions of other types in all the bones and joints, as osteoarthritis (tuberculous) of the hip, wrist, and knee; hypertrophic arthritis of the spine; fractures of all varieties; tumors, and finally periostitis and hyperostoses of the bones of the legs as a result of chronic

TABLE I: TYPES OF DISEASE

Lepromatous type		Neural type	
L1.....	51	N1.....	21
L2.....	41	N2.....	34
L3.....	10	N3.....	62
TOTAL.....	102	TOTAL.....	117
Mixed cases			
L1-N1.....	38		
L1-N2.....	25		
L1-N3.....	36		
L2-N1.....	38		
L2-N2.....	50		
L2-N3.....	38		
L3-N1.....	5		
L3-N2.....	16		
L3-N3.....	18		
TOTAL.....	264		

irritation in patients with trophic cutaneous ulcers, which, by the way, were frequent. We also found pulmonary tuberculosis, gastric carcinoma, and other diseases in association with leprosy."

It was decided, in view of Dr. Acosta's conclusions, confirming those previously published, to limit our study to the bones of the hands and feet. Films were obtained of 532 persons—483 with leprosy, 5 suspected of having the disease, and 44 in normal health. Films of the hands numbered 469, of the feet 372.

Among the 483 lepers examined, all variations of the disease were found. Because of this, it may be well to explain the two classifications kept in view throughout our study, one scientific and the other administrative.

According to the specifications of the International Congress of Leprosy at Cairo (1938), there are two types of leprosy: (1) the nerve or neural type (denoted by N), including all cases of benign leprosy with polyneuritic manifestations (disorders of sensation, trophic changes,

<sup>1</sup> Presented at the Second Inter-American Congress of Radiology, Habana, Cuba, Nov. 18, 1946. Accepted for publication in June 1947.

paralyses, mutilations, etc.), or macules of non-leprous nature ("leprides"), or both; (2) the lepromatous type, formerly called cutaneous (denoted by L), in which are grouped all cases of malignant leprosy with poor resistance and unfavorable prognosis, with lesions especially on the skin and other organs, and in general showing polyneuritic manifestations only when they are very far advanced or when they have a tendency to become neural secondarily.

These two types are subdivided according to the degree of involvement: first degree neural (N1), moderate neural (N2), advanced neural (N3), first degree lepromatous (L1), moderate lepromatous (L2), and advanced lepromatous (L3).

When the cases are of mixed type, as occurs in most instances, we denominate them with the letters L and N, followed by a number, to indicate the extent of the lesions. There are thus nine type of mixed leprosy, as follows: L1-N1, L1-N2, L1-N3, L2-N1, L2-N2, L2-N3, L3-N1, L3-N2, L3-N3.

In the administrative classification, according to Colombian law, we have to consider (1) patients with active lesions; (2)

TABLE II: ADMINISTRATIVE CLASSIFICATION

Active cases.....	393
Improved under observation.....	19
Social cures.....	6
Relapsed social cures.....	21
Handicapped social cures.....	44
Suspicious under observation.....	5
Cured.....	44
TOTAL.....	532

those under observation as improved; (3) those who, after a period of observation and clinical and laboratory examinations confirming the absence of active lesions, are known as "social cures"; (4) those who may be regarded as cured but whose lesions have deprived them of the possibility of a normal life; and (5) those who, after a social "cure" have had a relapse and are classified as "relapsed social cures."

According to the previous classifications, and taking into consideration the age and sex, our 532 observations may be grouped in the accompanying tables.

TABLE III: CLASSIFICATION BY AGE AND SEX

Age Group (Years)	Ill	Suspicious	Healthy
2-10	14	..	10
11-20	46	3	13
21-30	81	1	7
31-40	109	1	10
41-50	107	..	2
51-60	83	..	2
61-70	33	..	..
71-80	6	..	..
81-90	4	..	..
Males (391)	365	2	24
Females (141)	118	3	20
TOTAL	483	5	44

## DECALCIFICATION AND RAREFACTION

Side by side with the leprous mutilations caused by trophic lesions, described by all investigators, our attention was called from the start to the decreased density and great transparency of the bones in many of the patients examined. In neural leprosy, as well as in the lepromatous and mixed cases, there is frequently found in the epiphyses of the phalanges, metacarpals, and metatarsals, a trabecular structure much more differentiated than in healthy individuals (Fig. 1). At the same time the cortical covering of the diaphysis is thinner and the medullary canal is widened (Fig. 9). The loss of calcium salts, to which the prominence of the trabecular structure is attributable, precedes the rupture of some of the trabeculae and results in the formation of clear circumscribed zones, located in general at both ends of the phalanges and the distal ends of the metacarpals and metatarsals. Such vacuoles do not determine any peripheral reaction, but when they approach the cortical covering they distend it, sometimes destroying it. By a similar process the nutrient foramina are enlarged, so that they often become visible on the x-ray film, as described by Murdock and Hutter, who regard such enlargement as a response to the pathologic leprous vascular supply (Fig. 2).

These evidences of decalcification and rarefaction, sometimes producing a true vacuolar picture, may be found at any age and in all types of leprosy (Fig. 3). In several children in whom ossification was

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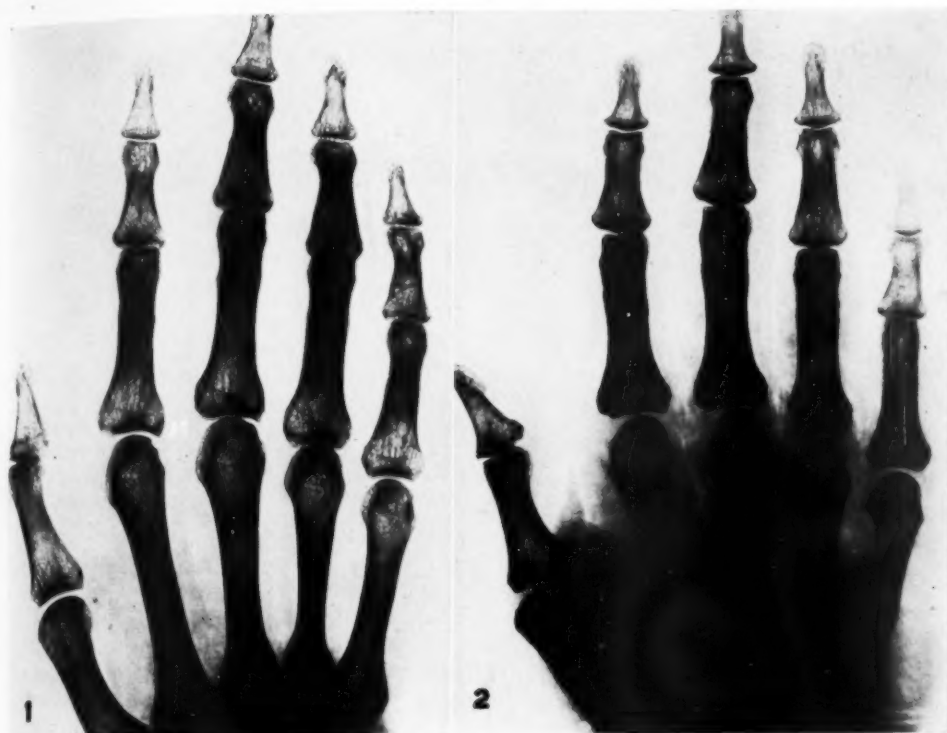


Fig. 1. Right hand, showing osseous rarefaction, especially noticeable at the epiphyses. Male, age 46; duration of disease 16 years; type L2-N1, active; blood calcium 0.02.

Fig. 2. Right hand, showing enlargement of the nutrient foramina in the first and second phalanges of the third and fourth fingers. Male, age 70; duration of disease 6 years; type L2-N1, active.

far from complete, we were able to observe decalcification, rarefaction, and, occasionally, vacuolar appearances. Such manifestations, however, are more apparent in the bones of the hands than of the feet.

These vacuolar rarefactions, we believe, are due in some instances to nerve damage and in others to the growth of Hansen's bacilli in the bone marrow and in the osseous tissue. Only in this manner were we able to explain the presence of this finding not only in the neural type of the disease but also in the lepromatous type and in predominantly lepromatous mixed types.

As to the decalcification and the first phase of osseous rarefaction, we may mention three factors to explain the mechanism of their production: impaired circulation as a result of deterioration of the sympathetic fibers of the nutrient arterioles;

disorders of the nerve supply due to the poor condition of the conductive nerves; and disturbed metabolism of calcium salts. The first two factors are well known and generally accepted. As to the last one, the results of examinations carried out up to now are contradictory: out of 85 determinations of blood calcium in the cases upon which the present paper is based, 6 were normal, while in the remaining 79 there was hypercalcemia, from 0.012 up to 0.028. Of 10 cases cited by Prof. José Ignacio Chala, one of the best known Colombian authorities on leprosy, 9 showed a hypercalcemia of over 0.017 and only one a normal figure (0.010). Dr. Hernando Groot, another distinguished Colombian investigator, on the other hand, in a study of 45 patients, found normal figures in the early cases and hypocalcemia when the disease was more advanced.



Fig. 3. Left hand, with vacuolar picture at the epiphyses of the first two rows of phalanges of the four inner fingers; arthritis between the two distal phalanges of the forefinger. Female, age 53; duration of disease 11 years; type L2-N1, active.

[Legend continued on opposite page]

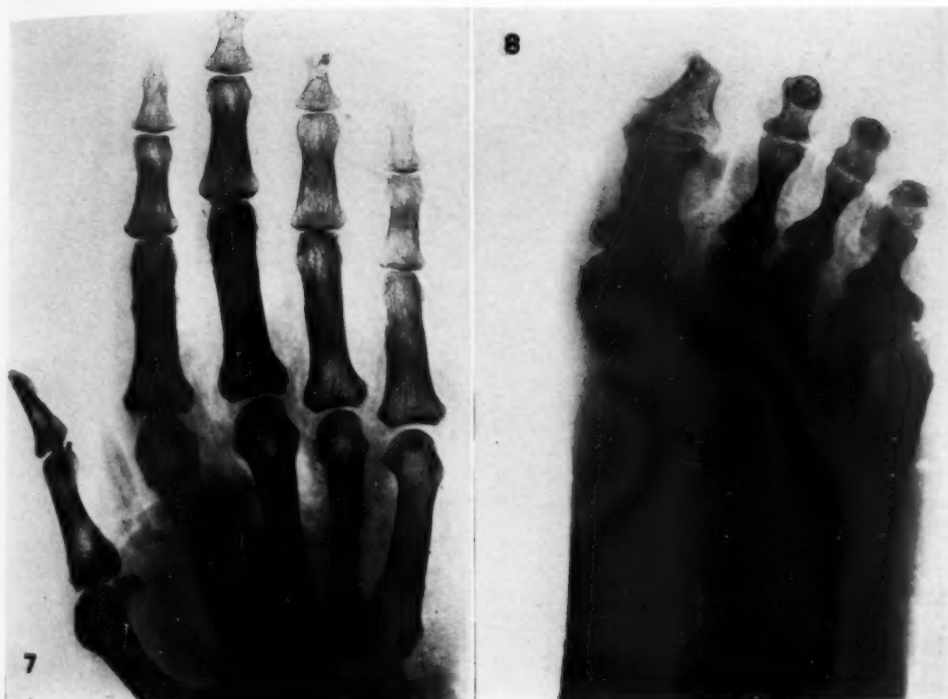


Fig. 7. Right hand: transverse section due to reabsorption in the distal end of the distal phalanx of the fourth finger; metacarpal-phalangeal rarefaction alternating with hypercalcification. Male, age 68; duration of disease 17 years; type L2-N1, active; blood calcium 0.009.

Fig. 8. Right foot: oblique section due to reabsorption in the distal end of the distal phalanx of the first toe; hypertrophy of the outer sesamoid of the first metatarsal; disappearance of the phalanges of the fifth toe, due to atrophy; union of fourth and fifth metatarsals and enlargement of the first phalanx of the fourth metatarsal at the proximal end. Male, age 50; duration of disease 30 years; type L2-N2, active.

Obviously rarefaction and decalcification are among the earliest radiologic findings in leprosy. But it is clear, also, that in the neural type of the disease and in the mixed types with preponderance of nerve lesions, most of the bones examined have regained their normal density. And since, quite without relationship to the clinical findings, roentgen studies have revealed hypertrophy and hypercalcification of the bones in some instances and in others have shown rarefaction of some bones and hypercalcification of those adjoining, it appears to us that the contradictory figures

as to the blood calcium are to be explained by the variety of the cases examined.

#### HYPERTROPHY AND HYPEROSTOSIS

As has just been stated, side by side with areas of rarefaction there may appear zones of hypercalcification occasionally accompanied by a noticeable widening of the proximal ends of one or more phalanges, covering the adjoining phalanx like a cap (Fig. 4). This appearance, called by one of us *en capuchón* (hooded), we consider typical of the disease because it appears not only in cases without mutilations, as in

Fig. 4. Right hand, showing "hood" picture (*en capuchón*), with enlargement and hypercalcification without atrophy, at the two distal phalanges of the third and fourth fingers. Male, age 29; duration of disease 3 years, type L1-N1, active; blood calcium 0.019.

Fig. 5. Left foot, with perforating ulcer; phalangeal reabsorption in the first and fifth toes. Another type of "hood" picture is shown. Male, age 40; duration of disease 6 years; type L2-N3, active.

Fig. 6. Right hand. Note the hypertrophy of the distal end of the second metacarpal and the "hood" picture at the distal phalanx of the thumb. Male, age 42; duration of disease 27 years; type N3, handicapped social cure.

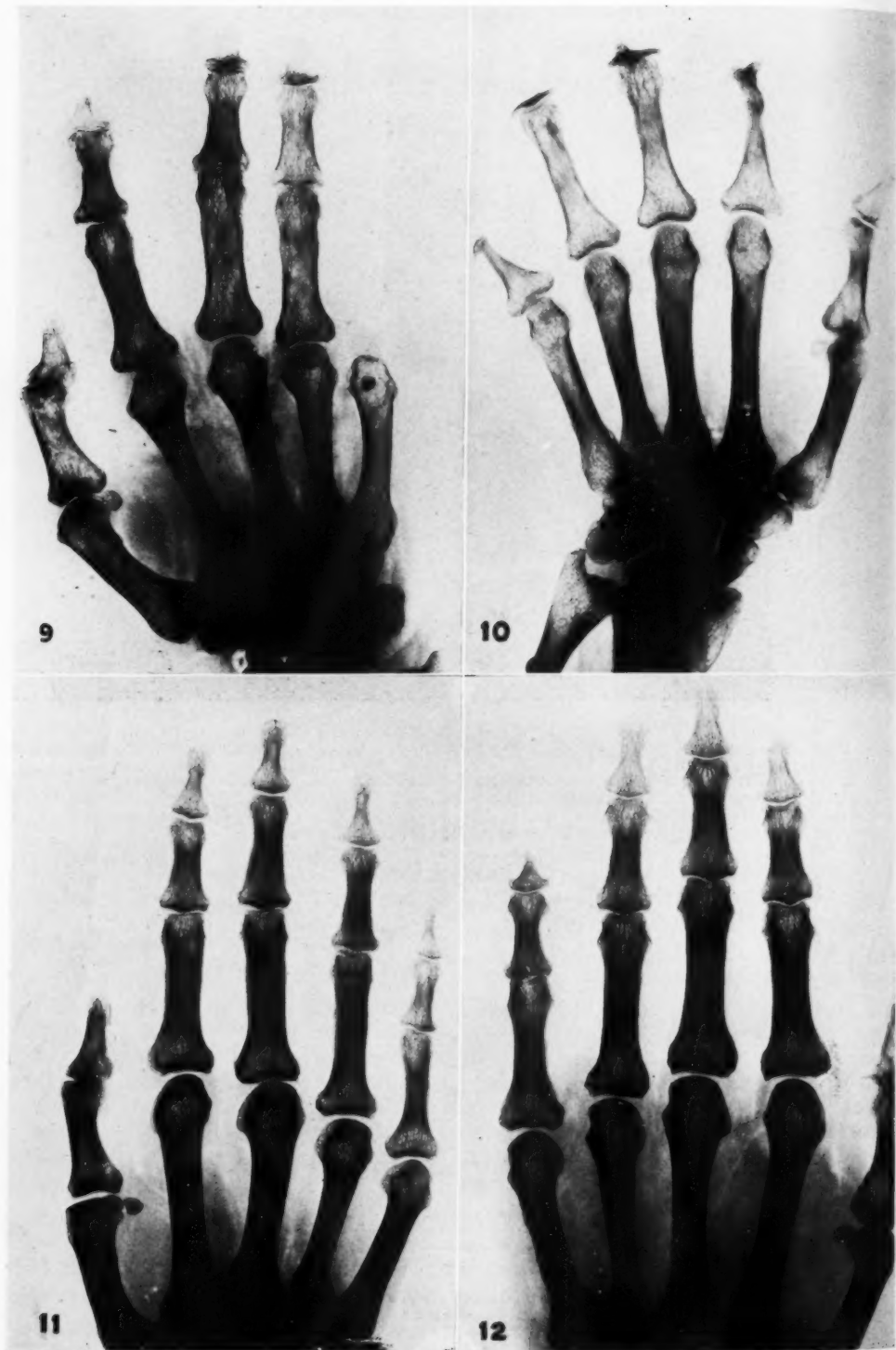


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Fig. 13. Left hand, with "collar-button" picture on distal phalanx of fifth finger and "hood" picture on distal phalanx of middle finger. Female, age 29; duration of disease 22 years; type L2-N2, active.

Fig. 14. Right foot, with "diabolo" [hour-glass] picture on the first phalanx of the fifth toe; reabsorption in all phalanges of the fourth and in the two distal phalanges of the fifth toe. Female, age 37; duration of disease 8 years; type N1, active.

the one illustrated in Figure 4, but also with different radiological characteristics in the rest of the phalanx in cases of neural leprosy with mutilations.

Hyperostosis occurs, however, without hypertrophy of the bone in many cases. Radiologically, we observe an increased density of phalanges, metacarpals, and metatarsals, especially at the diaphyses, contrasting with rarefaction of some phalanges and normal calcification of others (Fig. 5).

Similarly, there may be hypertrophy

without hyperostosis, at the articular ends of the phalanges and metacarpals, and particularly of the metatarsals, playing a very important part in certain joint deformities to be mentioned later (Fig. 6).

#### REABSORPTION AND ATROPHY

Leprous reabsorption takes two forms: simple reabsorption, and reabsorption following atrophy. Both are present in cases of pure neural leprosy of grades N2 and N3, and in the mixed leproses in which the neural component is of either of these

Fig. 9. Right hand, showing reabsorption of the distal phalanges of the three middle fingers and of all phalanges of the fifth finger. Note also the thinning of the cortical covering of the phalangeal diaphyses, with enlargement of the medullary canal. Female, age 63; duration of disease 30 years; type L3-N2, active.

Fig. 10. Left hand, showing "hood" picture in second phalanges, with reabsorption of first phalanges and of the rest of the second phalanges. The density in what remains is increased. The first and second phalanges of the forefinger are fused. Female, age 76; duration of disease 14 years; type N3, handicapped social cure.

Fig. 11. Right hand, with thinning, due to atrophy, of the distal phalanges of the fourth and fifth fingers, especially noticeable in the latter. Male, age 30; duration of disease 6 years; type L2-N1, active; blood calcium 0.013.

Fig. 12. Left hand: "collar-button" picture on distal phalanx of fifth finger. Male, age 46; duration of disease 23 years; type L2-N1, active.



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grades. Both forms affect the phalanges, metacarpals, and metatarsals, being more frequent in the distal phalanges, where the process generally begins. Finally, unlike disorders of sensation, reabsorption does not always start in the inner fingers of the hands, nor in the outer toes, for we have found several cases with early and more marked lesions on the distal phalanges of the three middle digits.

Simple reabsorption is revealed radiologically as an osteitis occurring at the distal ends of the third phalanges of hands and feet, without an adjacent hypertrophic reaction and without formation of sequestra. Sometimes, small erosions are to be seen on the terminal part of the phalanx; at other times the phalanx appears to be cut across, transversely or obliquely (Figs. 7 and 8). This type of simple absorption of the ends of the phalanges almost always begins with onychia.

The process does not always stop there, however, but seeks to invade the proximal end of the phalanx (Fig. 9). In this connection we wish to emphasize the defensive reaction opposed by the extremity to the advance of the process. The density first increases, and the end of the phalanx expands, thus producing another type of "hood" picture, similar to the one formerly described (Fig. 10). In spite of this presumably defensive reaction, the bone is eventually destroyed and the joint is involved.

Reabsorption following atrophy is a different process, because, like leprosy neuritis, with which it is associated, it appears in the areas belonging to the ulnar nerve in the hand and to the external sciatic nerve in the foot, and, as a rule, the more advanced the neural damage the more marked does the reabsorption be-

come. When it appears in the distal phalanges, the diaphysis begins to thin in its distal half, as happens on sharpening a pencil or sucking a stick of candy (Figs. 5, 11, and 17). This conical thinning slowly shortens the phalanx, with gradual disappearance of the distal portion, until only the proximal third is left, with a para-articular zone of normal thickness, the rest being very thin. At this time the appearance is that of a "collar-button," as already pointed out by other investigators. However, the process does not stop here, but advances until there remains only the para-articular portion unthinned, again producing the "hood" image (Figs. 12 and 13).

When the process begins in the phalanges of the first and second rows, the radiological appearance is different: the phalanx in which the cortical layer has previously thinned begins to narrow at the middle, until it takes as a whole the form of a "diabolo"<sup>2</sup> (Figs. 14 and 17), and as the atrophy continues in proportion to the course of the disease, a time comes when the phalanx fractures in the middle (Fig. 15). The distal end then disappears, owing to reabsorption, and the proximal portion assumes first a "collar-button" and later a para-articular "hood" appearance.

When in the reabsorption following atrophy, the lesion is located on the phalanges of the first two rows, those of the third row lose their normal articulation and are deflected, showing excessive distortions, resulting in muscular atrophy, tendinous retraction, and relaxation of the fibro-articular elements.

Finally, we must point out the fact that in several cases in which there has been re-

<sup>2</sup> Or an hour-glass.—ED.

Fig. 15. Right foot, with fracture, due to reabsorption, in the first phalanx of the second toe; atrophy of the distal phalanges of the third and fourth toes; metatarsal-phalangeal subluxation at fifth toe, and periostitis of the fifth metatarsal and first phalanx, caused by perforating plantar ulcer. Female, age 40; duration of disease 22 years; type L3-N3, active.

Fig. 16. Right hand, with advanced atrophy of the phalanges: "hood" pictures. Female, age 38; duration of disease 22 years; type L2-N3, active.

Fig. 17. Left foot, with advanced trophic lesions of the metatarsals and phalanges, subluxations, "collar-button," "diabolo" [hour-glass], and "stick of candy" pictures. Female, age 32; duration of disease 7 years; type L1-N2, active.

Fig. 18. Right foot. Atrophy of the phalanges has nearly reached the tarsus. Hammer foot. Male, age 46; duration of disease 15 years; type N3, active.

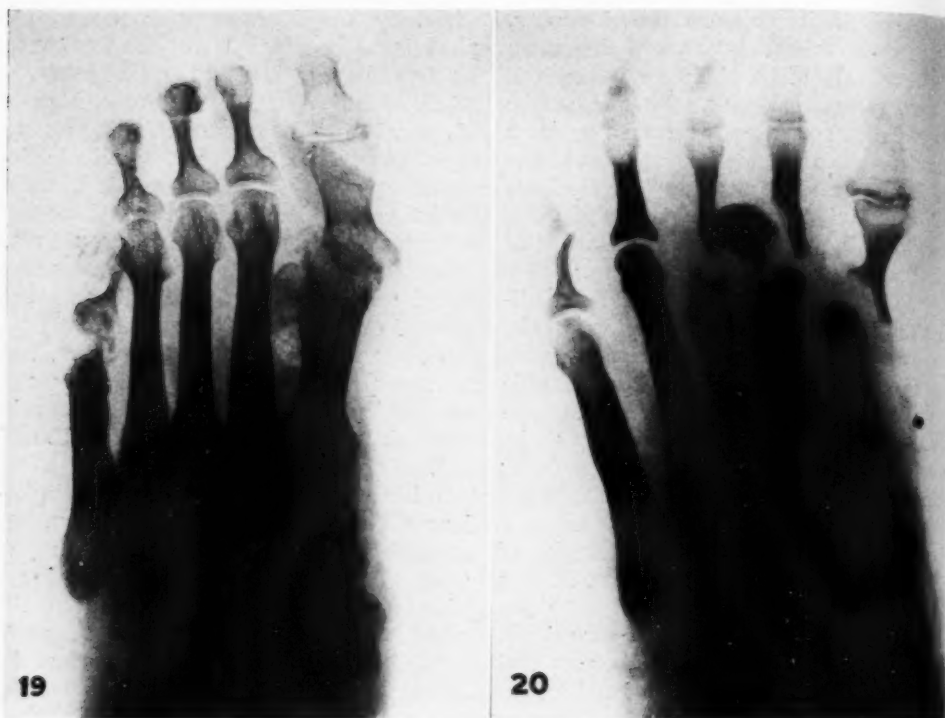


Fig. 19. Left foot, with perforating plantar ulcers at the anterior inner and outer arches. Male, age 25; duration of disease 6 years; type L2-N3, active.

Fig. 20. Left foot, with multiple perforating plantar ulcers of long duration. Female, age 34; duration of disease 8 years; type L1-N3, active.

absorption or atrophy of the metacarpals or metatarsals, as well as of the first row of phalanges, a synostosis is observed between the remaining phalangeal fragments and the atrophic ends of the metacarpals or metatarsals (Fig. 10).

#### MUTILATIONS

The trophic lesions already described, together with the loss of sensibility, cutaneous and vascular alterations, and progressive amyotrophy, transform the hands and feet of the leper into stumps of a truly pitiful appearance. These are the mutilations that accompany neural leprosy and the mixed type with neural predominance, in the last stage. Often they are observed in patients without Hansen's bacillus, and in those who, from a radiological point of view, no longer show decalcification and rarefaction.

Mutilations may affect the four extremities and are generally symmetrical, even if predominant in the lower limbs (Fig. 16 and 17). In the hand, the reabsorption stops at the carpus, while in the foot it stops at Lisfranc's articulation (Fig. 18), although on exceptional occasions it reaches the cuboid. In the hand, the destructive process is less noticeable on the outer portion; in the foot, the injury lessens in intensity from the outer edge to the inner and there is often found the appearance which Jeanselme has described as triangular.

#### PERFORATING PLANTAR ULCER

In spite of all that has been written on the radiology of leprosy, no attention has been given to the very characteristic radiological pictures of perforating plantar ulcer. This is a cutaneous trophic lesion

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Fig. 21. Right foot with analgesic whitlow in fourth toe. Male, age 45; duration of disease 10 years; Type N2, social cure.

Fig. 22. Left foot, with healed fracture of fifth metatarsal; trophic lesions of phalanges. Male, age 49; duration of disease 11 years; type N3, active.

located on the sole of the foot, especially on the anterior, inner arch, which through secondary infection causes in its final period a metatarso-phalangeal osteo-arthritis. The radiological picture is that of osteomyelitis, but the epiphyseal location, nearly always at the first metatarsal and adjoining phalanx, and the destruction of joint tissues as seen on the x-ray film, readily suggest a leprosy etiology. Healing takes place in most cases through ankylosis, with more or less marked deviations of the phalanges and metatarsals (Figs. 19, 20, and 24).

#### ANALGESIC WHITLOW

Leprous whitlow is a true osteomyelitis occurring with relative frequency especially in cases of neural leprosy and in the mixed cases with neural predominance. Radiologically it has the same aspect as osteomyelitis, but clinically it differs from the

latter in the absence of pain during the early phases of the disease (Fig. 21).

#### FRACTURES

Fractures are frequent in leprosy, not only as a result of reabsorption and atrophy but also of trauma to normal or decalcified bones. The traumatic fractures unite with formation of callus opaque to x-rays, as we could observe in three of our patients (Fig. 22). We differ, therefore, with those who maintain that fractures do not unite in lepers.

#### LESIONS OF THE JOINTS

Perforating plantar ulcers cause infectious arthritis just as whitlows do. This is one mode of joint attack in leprosy. A second form is due to reabsorption and atrophy. In such cases there is an osteoarthrosis, which may be of the hypertrophic type when there has been a growth of

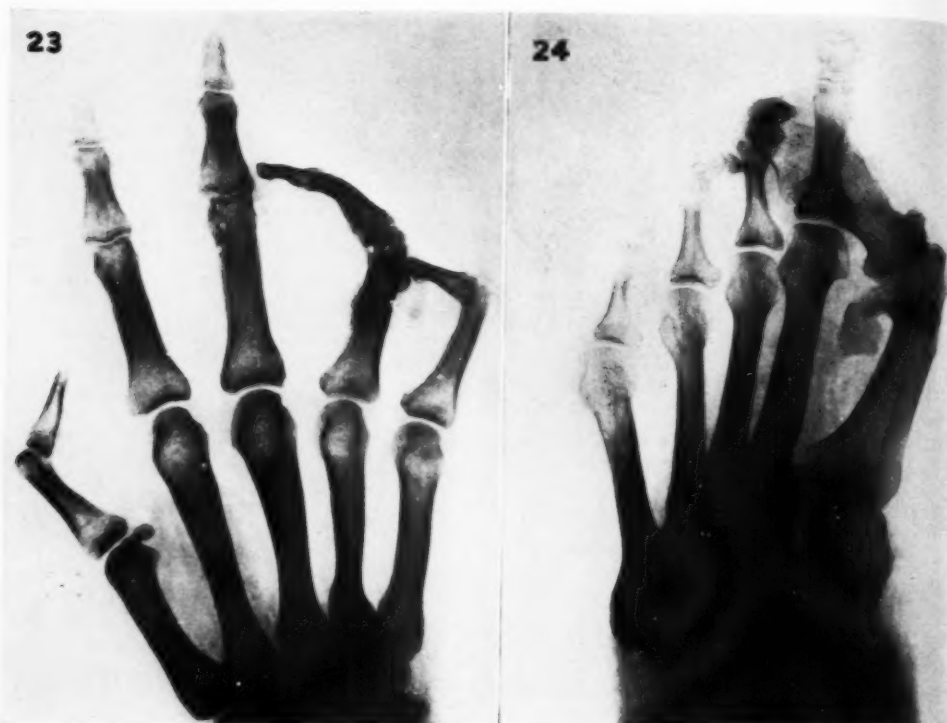


Fig. 23. Right hand, with retraction and hyperflexion of distal phalanges of first, fourth, and fifth fingers. Male, age 58; duration of disease 21 years; type L3-N3, active.

Fig. 24. Left foot, showing hallus valgus as a result of healing of a perforating plantar ulcer at the anterior inner arch. Male, age 40; duration of disease 15 years; type N3, social cure.

the joint ends, or of the atrophic type when there is a thinning of the latter through leprous reabsorption.

In either, the position of the bones forming the involved joint is altered, and more or less pronounced deformities appear (Figs. 23 and 24). The "claw hand" (*en garra*) caused by amyotrophy of the eminences or of the interosseus, with predominance of the flexor over the extensor muscles, and the "gale hand" (*en vendaval*), with hypertrophy of the osseous extremities and great deviations of the phalanges, are two examples of such deformities.

#### OSSIFICATION

In 46 healthy children of lepers and in 12 with the disease, all under eighteen years of age and over two, we failed to find any difference in the time of appearance of the epiphyseal ossific centers, as compared with

that for a group of normal children studied by one of us in Bogotá.

#### STATISTICAL OBSERVATIONS

Among 483 lepers examined, there were 306 with clinical evidence of involvement of the bones of the hands and feet, as retraction, reabsorption, mutilations, perforating plantar ulcers, and whitlows. As would be expected, in all these cases osseous changes were demonstrated radiologically. In the remaining 177 cases, the only signs of the disease were skin lesions, amyotrophies, or early retraction without clinical changes suggesting skeletal involvement. In 68 per cent of this latter group, however, roentgen studies showed bone changes suggestive of a leprous origin, namely, decalcification in 63 (36 per cent), rarefaction in 28 (15 per cent), atrophy in 20 (11 per cent), and hypertrophy and hyperostoses in 5 each

(3 per cent). In only 32 per cent was a normal radiological picture obtained. According to the Cairo classification the distribution of the 177 patients was as follows:

L1...34	N1...9	
L2...29	N2...6	
L3...10	N3...4	
L1-N1...26	L2-N1...27	L1-N1...5
L1-N2...4	L2-N2...20	L3-N2...3
L1-N3...0	L2-N3...0	L3-N3...0

These data show the importance of radiological study of hands and feet in the diagnosis of leprosy, not only in individuals clinically suspected of having bone lesions, but also in those of normal appearance.

Clinica de Marly  
Bogotá, Colombia, S. A.

#### REFERENCES

- ACOSTA, EMILIO: Estudio radiológico de las lesiones osteo-articulares en la lepra. Thesis, Bogotá, 1942.
- ESGUERRA-GÓMEZ, GONZALO: La ossificación de la mano en la ciudad de Bogotá.
- FAGET, G. H., AND MAYORAL, A.: Bone Changes in Leprosy: A Clinical and Roentgenologic Study of 505 Cases. *Radiology* 42: 1-13, January 1944.
- JEANSELME, ED.: La lèpre. Paris, Gaston Doin et Cie, 1933.
- KARASEFF, J.: Aspect radiographique des manifestations ostéo-articulaires dans la lèpre. *J. de radiol. et d'électrol.* 20: 373-382, July 1936.
- LONDONO, GARCIA VICTOR: Lesiones tróficas de la lepra en los miembros inferiores y su tratamiento por la simpaticotomía lumbar. Thesis, Bogotá, 1940.
- MARINO, S. MIGUEL: Los trastornos sensitivos y tróficos en la lepra. Thesis, Bogotá, 1937.
- MURDOCK, J. R., AND HUTTER, H. J.: Leprosy: A Roentgenological Survey. *Am. J. Roentgenol.* 28: 598-621, November 1932.
- OBERDOERFFER, M. J., AND COLLIER, D. R.: Roentgenological Observations in Leprosy. *Am. J. Roentgenol.* 44: 386-395, September 1940.
- VIAMONTE CUERVO, J. M., ET AL.: Sobre las alteraciones óseas en la lepra. *Rev. leprol., dermat. y síf.* 1: 148-154, July 1944.

#### SUMARIO

##### Lesiones Oseas y Articulares en la Lepra

Este estudio roentgenológico de los huesos de las manos y los pies fué realizado en 483 leprosos que representaban todas las formas y grados de la enfermedad. Los hallazgos radiográficos comprendieron descalcificación y enrarecimiento, traducidos por proyección de la trama trabecular del hueso y, en algunos casos, por un verdadero molde vacuolar: hipertrofia e hiperostosis, bien asociadas o separadas, reabsorción ósea simple, traducida por osteítis, y reabsorción postatrófica. Típicos cuadros radiográficos son los denominados en capuchón, en botón de camisa y en diábolo.

Otras características de la enfermedad consisten en mutilaciones debidas a procesos destructivos en las manos y pies; úlceras perforadas de la planta del pie que dan un cuadro radiográfico de osteomielitis pero caracterizadas específicamente por su localización epifisaria y destrucción de los tejidos articulares; panadizo leproso, osteomielitis verdadera pero sin dolor; y fracturas, que pueden cicatrizar con formación de callo.

Entre los 483 leprosos estudiados, había 306 con signos clínicos de invasión de las

manos y pies, en forma de retracción, reabsorción, mutilaciones, úlceras plantares perforadas y panadizos. Según era de esperar, las alteraciones óseas eran observables radiográficamente en todos esos casos. En los 177 restantes, los únicos signos de la dolencia consistían en lesiones cutáneas, amiotrofias o retracción incipiente sin patología clínica que indicara invasión del esqueleto. En 68 por ciento de este último grupo, la radiografía reveló, sin embargo, alteraciones óseas de probable etiología leprosa: descalcificación en 63 (36 por ciento), rarefacción en 28 (15 por ciento), atrofia en 20 (11 por ciento), e hipertrofia e hiperostosis en 5 de cada una (3 por ciento). Sólo en 32 por ciento se observó un cuadro radiológico normal. Estos datos revelan la importancia del estudio radiológico de las manos y los pies en el diagnóstico de la lepra, no solamente en las personas en que se sospecha la presencia de lesiones óseas sino también en las de aspecto normal.

Nota: Este trabajo fué publicado en español en el Boletín de la Clínica Marly, Tomo 8, No. 4, diciembre de 1946.

## Hydronephrosis

### A Radiologic Classification Based on Anatomical Variations<sup>1</sup>

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**H**YDRONEPHROSIS is defined as dilatation of the drainage system of the kidney, *i.e.*, the calices, the infundibula, and the pelvis. Although this term etymologically does not express the pathological condi-

From the point of view of their mechanism, hydronephroses comprise two basic etiologic groups, according to whether or not they are caused by an obstruction. Where they are so produced, the obstruc-



Fig. 1. Non-obstructive hydronephrosis (congenital). Ureteral orifices wide.

Fig. 2. Pregnancy hydronephrosis (breech presentation).

tion *per se*, it is so universally accepted that its elimination might cause considerable confusion. In spite of this fact, we should like to recall the suggestion made by others in favor of the more descriptive designations "pyelectasis" and "caliectasis." In this paper, however, we shall mainly use the traditional term.

tion creates a hindrance to the free flow of urine. This in turn leads to back pressure, dilatation, parenchymal damage, and impairment of function, with ultimate complete loss of renal tissue. The hydronephroses without demonstrable obstruction (Figs. 1 and 2) include cases of congenital origin (analogous to Hirschsprung's

<sup>1</sup> From the Departments of Radiology and Surgery, Tufts College Medical School, Boston, Mass. Presented at the Thirty-third Annual Meeting of the Radiological Society of North America, Boston, Mass., Nov. 30-Dec. 5, 1947.



Fig. 3. A. Combined hydronephrosis due to carcinoma of bladder. B. Severe combined hydronephrosis due to adenocarcinoma of ureter. C. Internal hydronephrosis due to tuberculous stricture of ureter.

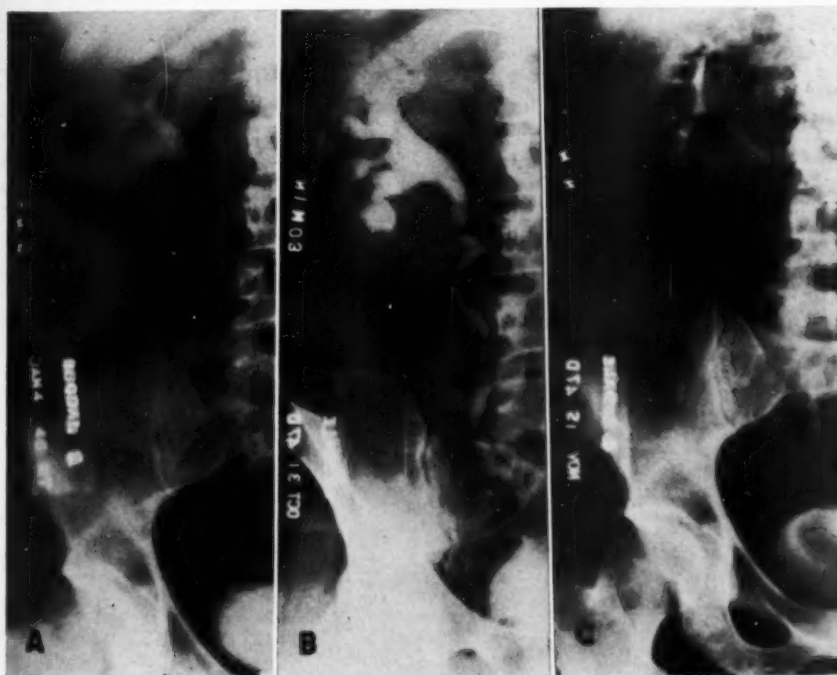


Fig. 4. A. Small stone in lower calyx. B. Combined hydronephrosis with stone in lower ureter, twenty-two months later. C. Marked improvement two days after ureteral catheter drainage.

disease), infection, and to some extent the important group of pregnancy hydronephrosis. There is much controversy as to the pathogenesis of the non-obstructive hydronephroses; various theoretical speculations account for them on the basis of toxic, hormonal, neurogenic, or neuromuscular factors. The great majority of hydronephroses can be identified as caused by obstruction, and therefore the present discussion is mainly based on that group.

The obstruction causing hydronephrosis may be due to either an intrinsic or an extrinsic cause, each of which in turn may be either congenital or acquired.

#### INTRINSIC CAUSES

##### *Congenital*

- Polycystic kidney
- Tumor
- Stone
- Kink
- Ureteral fold
- Ureteral valve
- Ureteral stricture
- Ureterocele
- Contracted vesical neck
- Urethral valve
- Urethral stricture

##### *Acquired*

- Stone
- Tumor
- Ureteral stricture
- Inflammation, infection
  - (a) non-tuberculous
  - (b) tuberculous
- Contracted vesical neck
- Urethral stricture
- Cyst and hypertrophy of verumontanum

#### EXTRINSIC CAUSES

##### *Congenital*

- Fused kidney; horseshoe kidney; cake kidney
- Aberrant vessel
- Abnormal insertion of ureter
- Abnormal rotation of kidney
- Ectopic kidney
- Double pelvis
- Double ureter
- Aneurysm of renal artery
- Tumor compressing urinary tract

##### *Acquired*

- Infection
- Adhesions, fibrous bands
- Tumor compressing urinary tract
- Dystopic kidney
- Ptosis
- Scoliosis
- Trauma (surgical or other injury)
- Hypertrophy of prostate gland

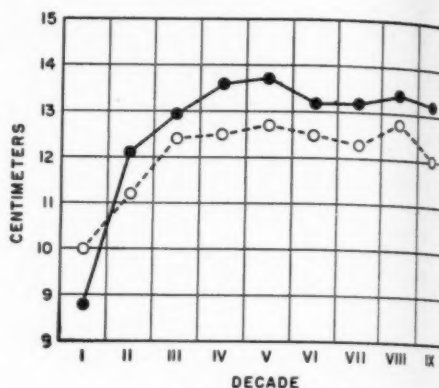


Chart I: Length of kidney

Solid line: male kidney. Average 12.6 cm  
Broken line: female kidney. Average 12.3 cm.

The obstructive process may occur at any level of the urinary tract. Early recognition of the condition and of the cause and site of the obstruction is essential for successful treatment and for prevention of permanent damage to the kidney (Figs. 3 and 4). The character of the dilatation of the pelvis, calices, or both, depends strictly on the type of the pelvis.

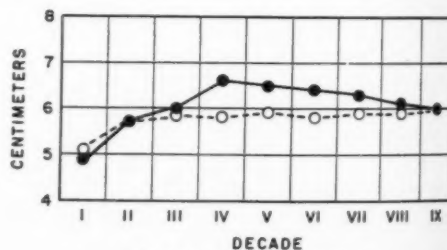


Chart II: Width of kidney

Solid line: male kidney. Average 5.65 cm.  
Broken line: female kidney. Average 5.77 cm.

Anatomically and roentgenologically there are three types of pelvis as regards their relationship to the kidney parenchyma; namely, intrarenal, intermediary, and extrarenal (Figs. 5 and 6).

For the purpose of this study 682 unselected roentgenograms representing 1,217 kidneys were analyzed. Measurements of the length and width of the kidney and the width of the parenchyma were taken. Of the 682 cases 54 per cent were in males and 46 per cent in females. A total of 61 cases

(9 per cent) showing hydronephrosis were not used for the normal measurements.

#### Average Measurements of Normal Kidneys

Length	
Males.....	12.6 cm.
Females.....	12.3 cm.
Width	
Males.....	5.65 cm.
Females.....	5.77 cm.
Width of parenchyma	
Males.....	2.2 cm.
Females.....	2.35 cm.

Analysis of the above figures revealed variations related to age and sex as shown in Charts I, II, and III, expressed in decades.

A study of the type of pelvis revealed the following percentage values.

#### Incidence of Different Types of Pelvis

Intrarenal	
Male.....	43.1%
Female.....	18.4%
Intermediary	
Male.....	56.3%
Female.....	77.3%
Extrarenal	
Male.....	0.6%
Female.....	4.3%

It will be seen that the intermediary type predominated and the intrarenal was second in frequency in both sexes, and that the incidence of the extrarenal type was extremely low.

The relative frequency of the intrarenal and intermediary types in each decade is shown graphically in Chart IV. The

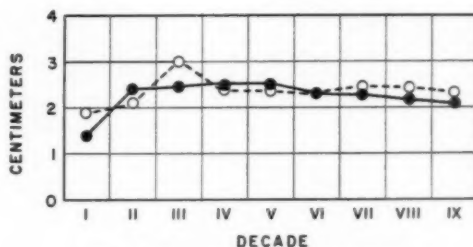


Chart III: Width of parenchyma  
Solid line: male kidney. Average 2.2 cm.  
Broken line: female kidney. Average 2.35 cm

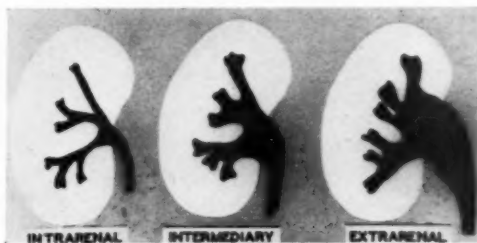


Fig. 5. Schematic drawing showing the three types of pelvis.

curves indicate the extremely low incidence of intrarenal pelvis in the early age groups—none in the first decade—and its continuous increase up to 50 per cent by the seventh decade. Correspondingly, the incidence of the intermediary type of pelvis in the first decade was almost 100 per cent, decreasing to 48 per cent by the seventh decade. These findings indicate that the renal pelvis changes its relationship to the kidney with increasing age, intruding into

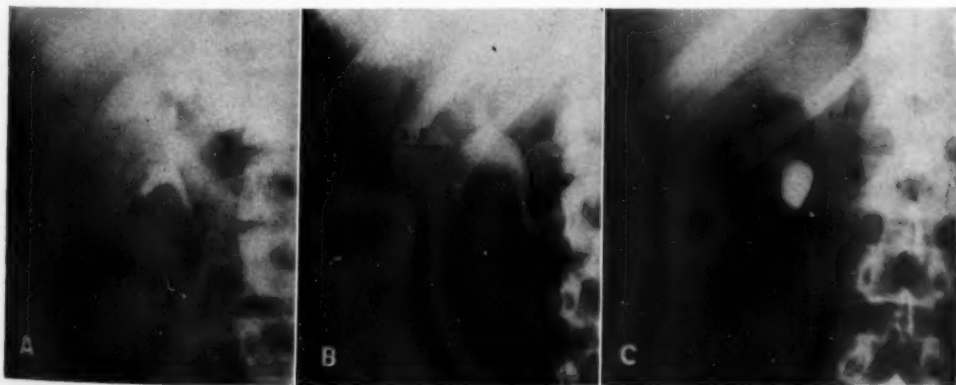


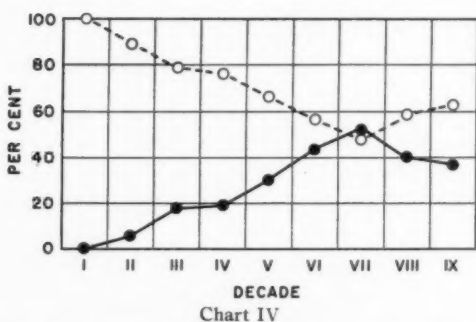
Fig. 6. Roentgenograms showing three types of pelvis: A. Intrarenal. B. Intermediary. C. Extrarenal.



Fig. 7. Bilateral internal hydronephrosis (due to stone). The width of the parenchyma on the right is 1.3 cm., on the left 0.9 cm.



Fig. 8. Severe combined hydronephrosis.



Solid line: percentage of intrarenal type pelvises (male and female)  
Broken line: percentage of intermediary type pelvises (male and female)

the parenchyma. Whether this phenomenon is due to fibrosis (sclerosis) of the kidney, the pelvis, or both, developing in older persons, which in turn tends gradually to pull the pelvis inward, remains to be investigated.

In 9 per cent of our cases we found bifid pelvises, 33 per cent of them on the right side and 67 per cent on the left. Double pelvises

occurred in 3 per cent of the series, also with 33 per cent on the right side and 67 per cent on the left. Sixty-three per cent were in males and 37 per cent in females.

Hydronephrosis being the most frequent manifestation of surgical renal disease, its importance must be duly realized by the roentgenologist. It should be borne in mind that hydronephrosis is not a disease *per se* but merely a sign. The conclusions drawn from its roentgen appearance as to site, type, and degree are of immeasurable diagnostic and pathognomonic value.

For evaluation of the changes in the urinary tract produced by obstruction, it is the function of the roentgenologist to impart a correct interpretation to the clinician in a precise manner. It has always been difficult to portray clearly to the urologist the extent of the pyelectasis and caliectasis present so that he can form in his mind the image displayed by the roentgenogram.

On the basis of anatomical considera-



Fig. 9. Extrarenal hydronephrosis due to aberrant vessel.

tions, we have classified hydronephrosis as internal, combined, and external, in a manner analogous to that used in classifying the type of normal pelvis; that is, according to its relationship to the kidney parenchyma. This classification illustrates clearly the localization of the dilatation. The roentgenologic appearance of each type is characteristic and throws light on the pathological anatomical process taking place in the kidney. The incidence of the three types in this series of 61 cases was as follows: internal, 31 per cent; combined, 46 per cent; external, 23 per cent.

The term "internal hydronephrosis" designates dilatation restricted to the intrarenal portion of the drainage system of the kidney (Fig. 7). It may involve all or any one of the calices, with or without dilatation of the pelvis. This is the type of hydronephrosis that occurs most frequently in intrarenal pelvises.

Combined hydronephrosis (Fig. 8) involves simultaneously both the calices and the pelvis, occurring most frequently in intermediary pelvises. It comprises the majority of all hydronephroses, which is to



Fig. 10. Width of parenchyma.

be expected in view of the fact that the largest number of pelvises are intermediary.

In external hydronephrosis (Fig. 9) the dilatation is restricted to the renal pelvis. It may occur in the extrarenal as well as the intermediary type of pelvis.

Hydronephrosis is a potential source of damage to the excreting tissue of the kidney. The increased pressure in the dilated structures exerts a deleterious effect on the adjacent cells, resulting in atrophy, directly proportional in degree to the character of the hydronephrosis and to its extent. The most severe damage to the parenchyma is brought about by the internal type of hydronephrosis. With the combined type the damage is less severe, and it is virtually non-existent with the external type, the effect of which is exerted centrifugally. The parenchyma in its entirety—that is, the medulla and cortex—is exposed to injury, resulting in impairment of function *via* the destroyed tubular and glomerular apparatus.

The width of the parenchyma as seen in the roentgenogram is an index of the degree of damage. It represents the distance between the tip of the calyx (fornix) and the nearest point of the outer border of the kidney (radius) (Fig. 10). Measurements taken from excretory urograms are more reliable than those taken from retrograde pyelograms, avoiding errors caused by artificial changes.

The figures showing the average width of the normal parenchyma given in Chart III

are results of measurements of our series, with the lowest reading of 1.45 cm. We consider it safe to state that any kidney parenchyma less than 1.45 cm. in width as measured on the roentgenogram is pathological.

#### SUMMARY

An analysis of the roentgenologic aspects of hydronephrosis (pyelectasis and caliectasis) is given, based on 682 roentgenograms representing 1,217 kidneys.

A classification of hydronephrosis is suggested according to anatomical variations. Measurements of the parenchyma prove to be a reliable index of the extent of damage resulting from hydronephrosis. In roentgenologic reports on urograms and pyelograms, statements as to the type of hydronephrosis and the measurements of the parenchyma are deemed to be of great value to the clinician.

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#### REFERENCES

1. ABESHOUSE, B. S.: Differential Diagnosis of Renal Neoplasms and Hydronephrosis or Pyonephrosis from a Pyelographic Standpoint. *Am. J. Roentgenol.* **45**: 214-220, 1941.
2. BIANCHI, M., AND GEISENDORF, W.: Diagnostic radiologique d'une hydronéphrose réservant quelques surprises. *Rev. méd. de la Suisse Rom.* **61**: 93-99, 1941.
3. DAVIS, D. M.: Ureteral Obstruction: Recent Advances in Its Embryology, Nosology, and Surgery. *Brit. J. Urol.* **19**: 71-82, 1947.
4. FUCHS, F.: Die Hydromechanik der Niere. *Ztschr. f. urol. Chir.* **33**: 1-144, 1931.
5. GIBSON, T. E.: Hydronephrosis: Classification and Plastic Repair of Ureteropelvic Obstructions. *Surg., Gynec. & Obst.* **80**: 485-496, 1945.
6. GRAUHAN, M.: Über Wachstum und Form der Hydronephrosen. *Arch. f. klin. Chir.* **180**: 517-539, 1934.
7. GREENE, L. F.: Renal and Ureteral Changes Induced by Dilating Ureter; Experimental Study. *J. Urol.* **52**: 505-521, 1944.
8. HINMAN, F.: Hydronephrosis; Structural Changes. *Surgery* **17**: 816-835, 1945.
9. HOGARTH, W. P.: Anuria Due to Large Hydronephrosis. *Canad. M. A. J.* **54**: 167-168, 1946.
10. HOSFORD, J. P.: Some Factors in the Causation of Hydronephrosis. *Lancet* **1**: 435-441, 1932.
11. ISRAEL, J.: Chirurgische Klinik der Nierenkrankheiten. Berlin, A. Hirschwald, 1901.
12. V. LICHTENBERG, A.: Die pathologische Physiologie der renalen Verstopfung und die sich daraus ergebenden konservativ-chirurgischen Massnahmen. *Acta chir. Scandinav.* **74**: 283-294, 1934.
13. MATHE, C. P.: Intrinsic Causes of Hydronephrosis. *J. Urol.* **38**: 574-592, 1937.
14. NARATH, P. A.: The Hydromechanics of the Calyx Renalis. *J. Urol.* **43**: 145-176, 1940.
15. ÖSTLING, K.: The Genesis of Hydronephrosis, Particularly with Regard to Changes at Ureteropelvic Junction. *Acta chir. Scandinav. (Supp. 72)* **86**: 1-122, 1942.
16. PAPIN, E.: Les hydronéphroses. Paris, Gaston Doin, 1930.
17. PEACOCK, A. H.: Hydronephrosis in Infants. *Northwest Med.* **43**: 110-112, 1943.
18. RAYER, P.: Traité des maladies des reins et des alterations de la sécrétion urinaire. Paris, J. B. Baillière, 1841.
19. STRONG, K. C.: Plastic Studies in Abnormal Renal Architecture. *Arch. Path.* **29**: 77-119, 1940.

#### SUMARIO

##### Hidronefrosis: Clasificación Radiológica Basada en las Variaciones Anatómicas

A base del estudio sistemático de 692 radiografías que representan 1,217 riñones, ofrécese un análisis de los aspectos radiológicos de la hidronefrosis (pielectasia y caliectasia). Las mediciones realizadas comprendieron el largo y ancho de los riñones y el ancho del parénquima.

Reconócense tres formas de pelvis renal: intrarrenal, intermedia y extrarrenal. En forma análoga clasifican las hidronefrosis en internas, combinadas y externas.

En la hidronefrosis interna, la más frecuente en las pelvis intrarrenales, la dilatación se limita a la porción intrarrenal del sistema excretorio del riñón. Puede afectar todos los cálices o cualquiera de ellos, con o sin dilatación de la pelvis. La

hidronefrosis combinada, que comprende la mayoría de los casos, afecta simultáneamente tanto los cálices como la pelvis, siendo más frecuente en las pelvis intermedias. En la forma externa, la dilatación se limita a la pelvis renal, pudiendo presentarse en las pelvis ya extrarrenales o intermedias.

Las mediciones del parénquima resultaron ser un índice fehaciente de la extensión de las lesiones debidas a la hidronefrosis. Puede considerarse como patológico todo parénquima renal que mida menos de 1.45 cm. de ancho. Una declaración relativa a la forma de la hidronefrosis y las mediciones del parénquima posee mucho valor para el clínico.

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# Effective Compression in Excretory Pyelography<sup>1</sup>

HERBERT R. ZATZKIN, M.D.

SINCE THE INTRODUCTION of intravenous pyelography by Swick (11) in 1929, the difficulty in obtaining uniformly good renal shadows has been recognized. Attempts have been made to remedy this situation.

Dehydration prior to pyelography is today a standard procedure and recently administration of pitressin for the concentration of diodrast has been suggested (12). The procedure of choice would be the administration of a drug to cause sphincteric contracture at the ureterovesical junction. Unfortunately such a drug is not known and other less efficacious means must be resorted to. Arendt and Maslow (1) fill the bladder with mineral oil and thereby prevent passage of the dye into the bladder. Council (4) places a balloon in the bladder. By this method both mechanical and physiological obstruction is produced, since the desire to void causes closure of the ureteral valves. These latter techniques nullify an important feature of excretory pyelography, its simplicity. Their practicality is questioned.

Most investigators have directed their attention toward producing obstruction of the lower ureters by abdominal compression, in an attempt to increase the concentration of contrast material in the proximal collecting system. A variety of homemade compression devices have appeared. Hudson (5) uses a small lint pad stuffed with cotton wool. Bell and his associates (2) employ a child's football or basketball bladder. Pressure is usually applied with a degree of firmness dependent upon the technician's estimate of what is needed to produce the desired effect. The patients' complaint of discomfort is used by some as indicating the point beyond which further pressure should not be applied. The reliability and accuracy of ure-

teral obstruction produced by such methods is at best questionable, and the need for further refinement of the technic is apparent.

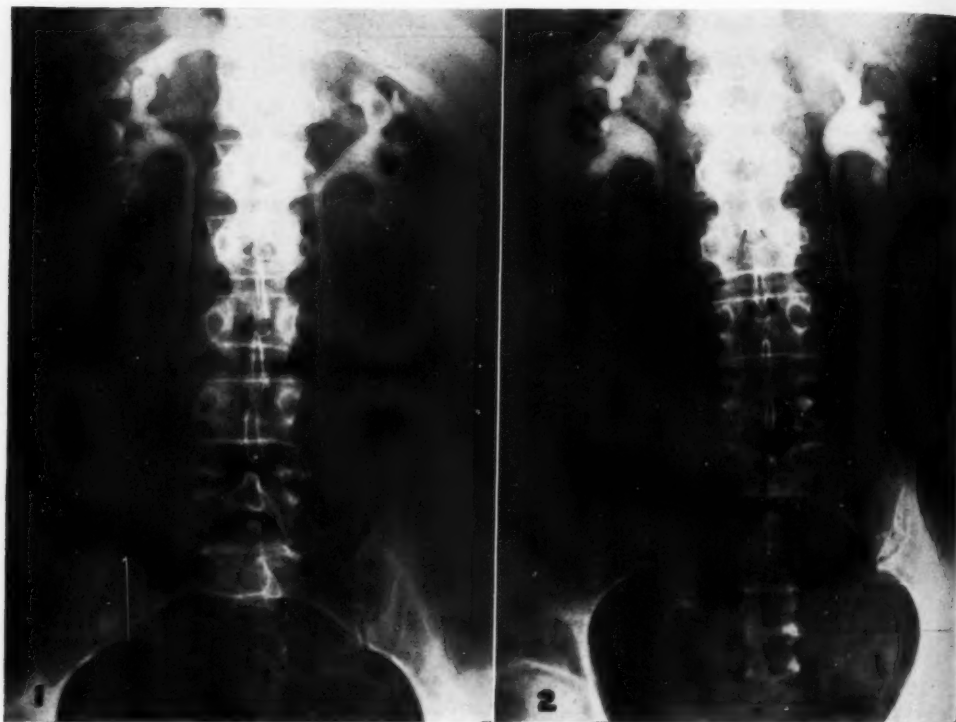
A simple and effective instrument for applying abdominal compression is being used by the Department of Roentgenology in conjunction with the Department of Urology at the University Hospital, Ann Arbor, Michigan. The procedure promises to eliminate much of the guesswork and cumbersomeness of the older methods. The cuff of a baumanometer is used as the compression device. It is placed beneath an abdominal binder and remains connected to the recording instrument by its tube, which extends from under the binder. The desired pressure can be applied at will by the technician.

## TECHNIC

The patient is dehydrated and all food is withheld eighteen hours prior to excretory pyelography. Though our own experience, as well as the work of Pearman (9), has indicated the superiority of castor oil in ridding the abdomen of gas, the use of this unpleasant laxative has not proved necessary. If, by the excretory method, renal sinus shadows are produced which are comparable in intensity to those obtainable by retrograde injection, the presence of some intestinal gas is not too objectionable. The routine administration of magnesium citrate the evening prior to examination has proved satisfactory in the majority of instances.

The procedure may be summarized briefly as follows: With the patient lying on his back on the urological table, the baumanometer cuff is placed under the binder. The cuff will fit nicely between the anterior superior iliac spines, approximately one inch above the pubis. The

<sup>1</sup> From the Department of Roentgenology, University of Michigan, Ann Arbor, Mich. Accepted for publication in August 1947.



Figs. 1 and 2. Individual cases with pressure device applied for ten minutes. Note sharp termination of ureters at point of obstruction. Though considerable gas is present in the bowel, visualization of pelves and calices is nevertheless good.

binder is tightened to its capacity, care being taken to prevent slipping of the cuff underneath. It is especially important that adequate pressure be exerted over the left ureter. Compression of the right ureter is more easily accomplished. This has been stressed by Rose (10), who points out that it may be due to interposition of the sigmoid colon. The tube leading from the pressure cuff is directed inferiorly but is not connected to the recording device until pressure is to be applied.

Four successive roentgen exposures are made, as follows:

1. A preliminary exposure is made, following which neo-iopax is introduced by vein. The injection should be done slowly, 30 c.c. in one full minute.

2. Five minutes later a second film is exposed. The rubber tube is then connected to the recording apparatus and pressure is immediately applied. A pres-

sure equal to 110 mm. of mercury will completely occlude the ureters in the majority of instances. In a stout person, or one of heavy musculature, 120 mm. of pressure may be necessary. Pressure beyond 120 mm. of mercury is poorly tolerated and for thin subjects 100 mm. of mercury will usually suffice.

3. Fifteen minutes after injection a third film is exposed with the pressure apparatus in place.

4. The patient is then placed in the upright position and the fourth exposure is begun no later than ten seconds following the release of pressure. This usually results in good visualization of the lower ureters. Speed is essential in making this exposure to avoid undue escape of opaque material from the upper urinary tract.

This technic is modified slightly in cases of hypertension. Pressure is applied following an exposure made three minutes

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after injection and is maintained for nine minutes, at which time (twelve minutes after injection) a second exposure is made. The experience of the author, as well as the work of Pearman (9), has indicated that in the presence of hypertension kidneys excrete the radiopaque medium very rapidly in the majority of cases. Almost all of it may be in the bladder within five minutes after injection.

This method has been found applicable to the average adult, though results are unpredictable in the very stout. Pressure is poorly tolerated by neurotic patients and it should not be attempted in patients with a history of recent laparotomy. Indeed, trial pressure with the hand upon the abdomen will furnish a good index as to the suitability of a given patient for the compression technic.

#### DISCUSSION

The use of compression in excretory pyelography poses two controversial questions: (1) Can the ureters actually be occluded by abdominal compression? (2) Assuming that occlusion is obtained, do hydronephrosis and hydroureter occur incident to the compression and interfere with interpretation of the films?

Marshall and Shanks (7) and Carlson (3) believe that it is impossible to occlude the ureters by abdominal compression. The work of Bell *et al.* (2), Rose (10), Pearman (9), and Hudson (5) offers proof to the contrary. Pyelographic evidence obtained (Figs. 1 and 2) with the technic described above would seem also to indicate that obstruction of the ureters is possible.

It is interesting to note that in certain instances the pressure was insufficient to produce total occlusion of the ureters and, though seepage occurred into the lower ureters and bladder, there was still excellent visualization of the proximal collecting system (Fig. 3). Carlson might interpret this fact as evidence of failure to occlude the ureters, though there can be no question that the distention of the calices and pelves, as well as of the proximal ureters, is of greater degree than one would



Fig. 3. The compression device had been applied for ten minutes prior to exposing this film. Though complete ureteral obstruction was not obtained, as evidenced by escape to the bladder, pelves and calices are well distended.

expect in routine pyelography without compression. Carlson's sweeping statement as to "The Proven Ineffectiveness of the Compression Bag in Intravenous Pyelography" must be taken with considerable reservation, considering that his series included but 20 cases and his methods were experimentally unsound. It is apparent that on assumption of an upright position the resultant tightening of the abdominal muscles will counteract the abdominal pressure that is applied, and one may reasonably expect some leakage into the bladder in certain instances. We have observed this to be the case and we advocate rapid exposure of the release film with the patient upright. This cannot be used as an argument against the use of compression, since it is the fifteen-minute film made in the horizontal position which is most informative.

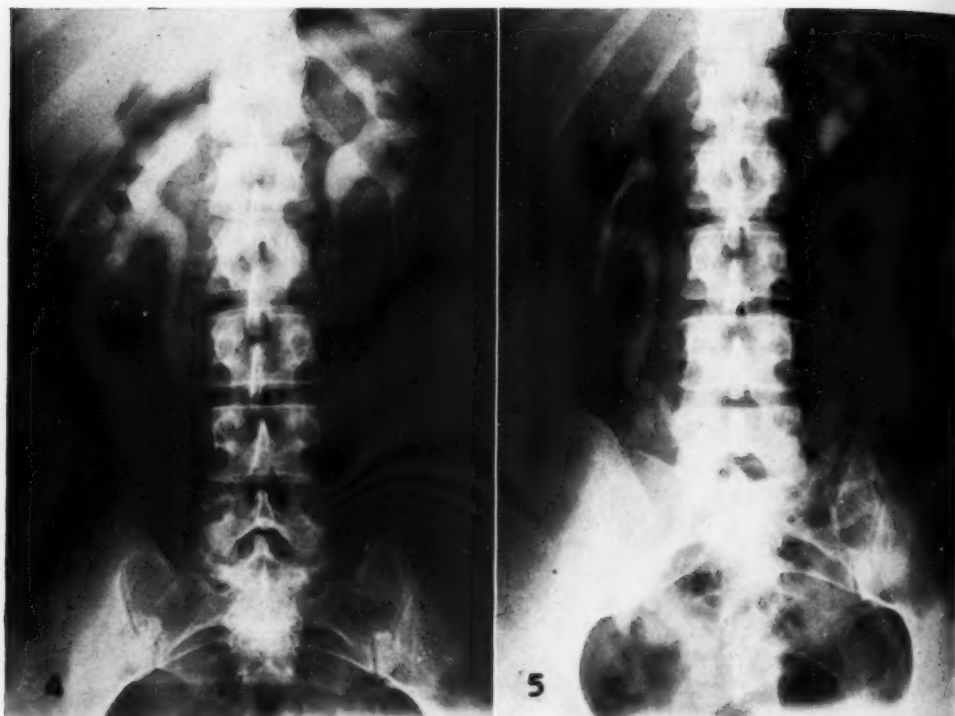


Fig. 4. Minimal hydronephrosis and hydroureter on the right. The pressure device is in place. The normal left side suggests that the dilated structures on the right are not merely attributable to compression.

Fig. 5. Same case thirty seconds following release of pressure, with patient in upright position. Persistence of dilatation on the right confirms the impression of hydronephrosis and hydroureter.

While some investigators are of the opinion that occlusion of the ureters by abdominal compression causes hydronephrosis and hydroureter (Figs. 4 and 5), the author has not found this to be the case, although he admits that a qualifying statement is indicated. That constant prolonged abdominal compression might induce hydronephrosis seems quite likely, though it is not within the scope of this paper to enter into that interesting aspect of the problem. It would seem unlikely that in the ten-minute interval during which pressure is applied, dilatation of renal sinuses beyond the limits of normal physiological distensibility would occur. The degree of distention observed (Fig. 7) is still within limits of normal. Nesbit (8) in a personal communication indicates that he does not believe that the dilatation demonstrated in such films is of sufficient

magnitude to interfere with their interpretation.

With the knowledge *a priori* that compression has been applied, it is unlikely that even the most inexperienced observer would interpret these dilated structures as indicative of pathology. Certainly one interprets retrograde films in the light of the knowledge that the structures are dilated; yet retrograde pyelograms are generally considered superior to excretory pyelograms in demonstrating anatomical details of renal sinuses. Abdominal compression with the baumanometer cuff would seem to give promise of eliminating the need for many retrograde examinations done solely to obtain a better view of renal sinuses.

Another point in favor of the procedure is the frequent demonstration of calices in different degrees of distensibility.

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Fig. 6. Five-minute film made without compression. The status of some of the calices and infundibula on the left is in doubt.



Fig. 7. Same case after pressure had been applied for ten minutes. All structures are within normal limits. Distention of pelvis and calices is not of such magnitude as to be interpreted as hydronephrotic.

Whereas narrowing of an infundibulum may be suggested on a five-minute film, seeing this structure well distended on a fifteen-minute film made with pressure eases the mind of the observer concerning scarring or other pathological change (Figs. 6 and 7).

#### SUMMARY

1. A simple effective compression device is described, whereby abdominal compression can be so controlled as to produce complete or partial ureteral obstruction.
2. The dilatation of renal sinuses is not of such magnitude as to interfere with interpretation.
3. Excretory pyelograms approaching in density those made by the retrograde method can thus be obtained.

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#### REFERENCES

1. ARENDT, J., AND MASLOW, L. A.: Blocking of the Ureters in Intravenous Pyelography by Filling the Bladder with Oil. *Radiology* **35**: 350-352, 1940.
2. BELL, J. C., HEUBLEIN, G. W., AND HAMMER, H. J.: Roentgen Examination of the Urinary Tract, with Special Reference to Methods of Examination and Findings in Individuals with Testicular Tumors. *Am. J. Roentgenol.* **53**: 527-562, 1945.
3. CARLSON, H. E.: The Proven Ineffectiveness of the Compression Bag in Intravenous Pyelography. *J. Urol.* **56**: 609-611, 1946.
4. COUNCILL, W. A. H.: A New Technique for Intravenous Urography. *J. Urol.* **46**: 143-145, 1941.
5. HUDSON, E.: Some Aspects of Compression in Intravenous Pyelography. *Radiography* **9**: 28-29, 1943.
6. JACHES, L., AND SUSSMAN, M. L.: Roentgenologic Diagnosis of Diseases of the Urinary Tract. In *Diagnostic Roentgenology*, Ross Golden, editor. New York, Thomas Nelson & Sons, 1941.
7. MARSHAL AND SHANKS: In a Text-Book of X-Ray Diagnosis, S. Cochrane Shanks, Peter Kerley, and E. W. Twining, editors. London, H. K. Lewis & Co., 1938, Vol. 1, part 3, p. 494.
8. NESBIT, R. M.: Personal communication.
9. PEARMAN, R. O.: Technical Considerations in Excretory Pyelography. *New England J. Med.* **228**: 507-508, 1943.

10. ROSE, J. A. G. F.: Compression Technique in Intravenous Urography. A Survey of 100 Cases. *Brit. J. Radiol.* 15: 266-268, 1942.

11. SWICK, M.: Intravenous Urography by Means

of Uroselectan. *Am. J. Surgery* 8: 405-414, 1930.

12. WALD, M. H., AND GALLOWAY, A. F.: Pituitrin for Concentrating Diodrast in Excretory Pyelography. *Radiology* 43: 358-363, 1944.

#### SUMARIO

#### Compresión Efectiva en la Pielografía Excretoria

Descríbese un procedimiento sencillo para producir compresión en los uréteres a fin de obtener una concentración mejor de colorante en la porción proximal del aparato colector al ejecutar una pielografía excretoria. Como compresor se utiliza el manguito de un baumanómetro, colocándolo debajo de una faja abdominal, y conectando con el registrador el tubo que sale de debajo de la misma. Se toman

radiografías antes y después de aplicar la presión. Para la completa oclusión de los uréteres suele bastar con una presión de 110 mm. de mercurio.

Con este sistema pueden obtenerse pielogramas excretorios que se comparan favorablemente en densidad con los retrógrados. La dilatación de los senos renales llenos de colorante no es tal que impida la interpretación de las películas.



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## Further Observations on the Use of Three-Million-Volt Roentgen Therapy<sup>1</sup>

RICHARD DRESSER, M.D.

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TEN YEARS AGO A million-volt x-ray generator developed by Van de Graaff, Trump, and their associates was put into operation at the Collis P. Huntington Memorial Hospital. This was the beginning of supervoltage x-ray therapy in Boston. A modification of this apparatus was later installed in the Massachusetts General Hospital. Six years ago the high-voltage research group at the Massachusetts Institute of Technology constructed an x-ray unit capable of running continuously at three million volts. This machine, located on the grounds of the Institute, was made available for therapeutic purposes, and a group of patients was treated. A preliminary report on this work was presented at the annual meeting of the American Roentgen Ray Society five years ago. The medical program was interrupted shortly after the entrance of the United States into the war, when it became necessary to turn the apparatus over to physical research. It was not until early in 1946 that the treatment of patients was resumed.

The therapeutic factors have been previously set forth, but are here briefly reviewed. The target-skin distance employed in the majority of cases has been 100 cm. Filtration is accomplished by aluminum, copper, lead, a thin layer of cooling water, and the gold inherent in the anode. This is the equivalent of approximately 20 mm. of lead. The intensity at 100 cm., with one-half milliamperere of current, ranges between 80 and 100 r per minute. The 10-cm. depth dose with small portals of entry is approximately double that obtained with 200 kv.; that is, a portal which would result in a 30

per cent depth dose with 200-kv. rays gives nearly a 60 per cent dose with three-million-volt rays. When large portals of entry are employed, the discrepancy in depth dose becomes less marked.

It has been pointed out that with supervoltage roentgen rays the point of maximum intensity is not on the surface of the skin, but slightly below it. This subcutaneous maximum intensity is the figure used in all expressions of dosage. It is common knowledge that with increase in voltage there is also an increase in the tolerance of the skin to radiation. An explanation of this will be set forth in the following paper, by Dr. Trump (p. 649). With three-million-volt rays it has been possible to deliver upwards of 5,000 r to a single portal, in the usual fractionation of 300 to 400 r daily, without visible skin erythema and without epilation of the pubic hair. This increase in skin tolerance, together with the increase in depth dose, is definitely advantageous in the treatment of deep-seated lesions.

In order to administer adequate depth doses with 200-kv. roentgen rays, it is frequently necessary to increase unduly the size of the portal of entry, thereby irradiating a considerable volume of normal tissue outside the tumor-bearing area. Since the size of the portal of entry has little effect on the depth dose of supervoltage roentgen rays, it is not necessary to resort to this expedient. Another means of augmenting the deep tumor dose is cross-firing. In the treatment of uterine cancer, for example, it is common practice to use three or four portals of entry, and some radiologists employ as many as six or eight. In the early work done at the

<sup>1</sup> Presented at the Thirty-third Annual Meeting of the Radiological Society of North America, Boston, Mass., Nov. 30-Dec. 5, 1947.

Massachusetts State Cancer Hospital on the combined x-ray and radium treatment of carcinoma of the cervix uteri, the author devised a simple mechanism for accurate direction of the x-ray beam. A small cylindrical piece of lead attached to a rod was pushed firmly against the cervix. A fluoroscopic screen was attached to the treatment couch directly below the patient, and a mirror was located at an angle which allowed visualization of the fluoroscopic image of the lead marker. It was the practice to center the patient as accurately as possible without the use of fluoroscopy and then to check the centering after the x-ray exposure was started. With portals of 100 to 150 sq. cm., it was quite surprising to find that, without the use of the fluoroscope, the lead marker was frequently not included in the field of radiation. In other words, the cross-fire treatment of a deep-seated tumor through small portals of entry without some means of accurately centering the beam, is likely to prove unsatisfactory. Ewing once remarked that it is the last 10 per cent of the radiation which effects a cure and not the first 90 per cent. When three-million-volt roentgen rays are employed, cross-fire irradiation is not necessary. The most advantageous portal of entry may be selected and the entire dose given through that portal. This eliminates critical angulation of the beam. The limitation of dosage is not the skin effect, but the tolerance of the underlying tissues.

A number of erroneous ideas regarding the absorption of supervoltage roentgen rays have become prevalent. This is perhaps a problem for the physicist, but it might be well to touch upon it from the clinical standpoint. Reducing the case to an absurdity, one would not treat a carcinoma of the cervix with x-rays produced at, let us say, 100 kv. with no filter and a focal skin distance of 20 cm. Nearly all the radiation would be absorbed in the first two or three centimeters of tissue, and a negligible amount would reach the cervix. However, these factors have been satisfactorily employed for years in the treat-

ment of carcinoma of the skin. Conversely, if one were treating a superficial epithelioma, it would be unwise to use three million volts with a filter of 20 mm. of lead and a focal skin distance of 100 cm. By the time the skin lesion had received an adequate dose, severe damage would have been done to the underlying structures.

The question of effect of the exit dose of supervoltage roentgen rays has frequently been brought up. In the cases of thoracic, abdominal, and pelvic cancer which we have treated through a single portal of entry no visible skin reaction has occurred on the side opposite that irradiated. However, cases of carcinoma of the mouth and metastatic cervical nodes treated through a single portal have presented epilation and slight erythema on the opposite side.

The use of supervoltage roentgen rays has met with varying degrees of enthusiasm. Holmes and Schulz, in a review of the work done at the Massachusetts General Hospital, have evinced some disappointment in their results. On the other hand, Sigv N. Bakke, radiologist at the Municipal Hospital, Bergen, Norway, has obtained quite a different impression. He has been treating patients since 1942 using an x-ray generator of the Van de Graaff type operating at a million and a half volts. Within the past year I had the pleasure of a visit from Dr. Bakke. A paper which he delivered at a local medical meeting has not been published, but he has given me permission to quote him freely. Bakke's supervoltage clinic at the time of his visit to this country averaged forty to fifty treatments per day. He is a radiologist of wide experience, and his judgment is not to be taken lightly. Bakke is certain that results have been obtained with supervoltage x-rays which have not been possible with any other type of radiation. Friedman is also convinced of the superiority of supervoltage therapy in certain cases.

The advent of supervoltage x-rays has been greeted by many with the same

skepticism which was accorded 200-kv. x-rays twenty-five years ago. This, of course, is not an unhealthy scientific attitude. In my early days of roentgen therapy in Baltimore, I recall that Baetjer, then roentgenologist at Johns Hopkins, opposed most vigorously the installation of a 200-kv. therapy unit in the hospital. He argued that sufficient damage was being done to superficial tissues with x-ray and that there was no need for extending this damage to deeper structures. The pioneer work in Baltimore in this field was left to Waters and Pearson, and to Howard Kelly. It was a number of years before "deep therapy" became generally accepted and, in fact, many years of experimentation were necessary before it was used to best advantage.

Statistical evaluations in the treatment of malignant disease are likely to be misleading. This applies to surgical as well as radiation results. Let us take for the example the treatment of carcinoma of the cervix uteri. The reports on the results of hysterectomy are usually based not on the entire group of cases, but on a selected few which are considered suitable for operation. On the other hand, radiation results are generally based on the treatment of all comers, both favorable and unfavorable. Every radiologist knows that a few cases which appear clinically hopeless at the outset will do exceedingly well and may even be permanently cured by adequate radiation methods. If the surgical figures were compiled from the entire group, both operable and inoperable, they would appear much worse than the radiation results. This does not mean, however, that surgery has no place in the treatment of carcinoma of the cervix. There is considerable discrepancy in the statistics on the surgical treatment of carcinoma of the breast. This does not reflect differences in surgical ability, but rather a difference in selection of cases. The man who always limits himself to the removal of growths well within the limits of operability will naturally report better results than one who is less conservative.

The radiologist must deal with a large number of patients who are considered hopeless from any other standpoint. He knows from experience that in many of these cases much palliation will be attained and that some may be cured. He has, however, no means of making a prognosis as to the response to radiation. It is largely a question of trial and error. In order that a few may be benefited, many must be subjected to a procedure which affords them little or no help. This state of affairs does not lend itself to impressive statistical results.

Every patient irradiated should be considered an individual problem. No stereotyped method or blanket form of treatment can be applied to all cases. We soon found, for example, that the super-voltage irradiation of breast tumors produced such undesirable results in the lungs that we reverted to the usual 200-kv. treatment. On the other hand, super-voltage x-rays, in our experience, present definite advantages in the treatment of deep-seated lesions. The employment of a single portal of entry is without doubt a step in advance. The fact that the size of this portal has no appreciable effect on the depth dose is exceedingly helpful in the treatment of well localized deep-seated lesions. The absence of skin reaction is a boon to those patients who are cured and who, if they had been irradiated at lower voltages, might develop late radiodermatitis. Striking examples of undesirable late skin changes in a series of irradiated pituitary tumors were recently shown by Sosman at a meeting of the New England Roentgen Ray Society. Presumably these changes could have been avoided if super-voltage radiation had been available.

We have frequently used three-million-volt x-rays to augment the dose in cases which have been previously irradiated with 200 kv., and in which the limit of skin tolerance has been reached. This has been particularly helpful in cases of carcinoma of the buccal cavity and metastatic cervical nodes. It should, however, again be emphasized that supervoltage radiation

must be used with caution for superficial lesions when there is danger of injury to underlying vital structures.

It has been our observation that the systemic reaction on the part of the patient is less when supervoltage x-rays are employed. This may well be accounted for by the fact that there is less scattering and that a relatively small volume of tissue is irradiated.

Commendation is due the high-voltage research group at the Massachusetts Institute of Technology, whose untiring efforts have made possible improvements in supervoltage apparatus. We are also greatly indebted to the Godfrey M. Hyams Trust, whose generous grants have furthered this work.

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#### SUMARIO

##### **Nuevas Observaciones sobre el Empleo de la Terapéutica de Tres Millones de Voltios**

Las observaciones descritas se refieren a la labor realizada con la unidad roentgenológica de tres millones de voltios, construida por el grupo de investigación de altos voltajes en el Instituto de Tecnología de Massachusetts.

La roentgenoterapia con tres millones de voltios ha mostrado ciertas ventajas sobre la irradiación con rayos generados a 200 kv. en el tratamiento de las lesiones profundas, y en particular mayor tolerancia cutánea y obtención de una dosis de mayor profundidad sin agrandar la puerta de entrada ni usar fuegos cruzados por distintas puertas. El punto de intensidad

máxima no queda en la superficie de la piel sino ligeramente debajo de ésta. La magnitud de la dosis no está, pues, limitada por la tolerancia de la piel, sino por la de los tejidos subyacentes.

La habitual distancia foco-piel es de 100 cm. Con aluminio, cobre, plomo, una delgada capa de agua refrigerante y el oro inherente en el ánodo, se obtiene una filtración equivalente a 20 mm. de plomo. A 100 cm., con medio miliamperio de corriente, la intensidad es de 80 a 100 r por minuto. La dosis a una profundidad de 10 cm. con pequeñas puertas de entrada viene a ser el doble que con rayos de 200 kv.



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# Physical Basis for the High Skin Tolerance of Supervoltage Roentgen Rays<sup>1</sup>

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THE PROGRESSIVE increase of skin tolerance with the quality of the incident roentgen radiation has been appreciated for many years in the lower voltage range (1, 2). Recent clinical observations with two- and three-million volt roentgen rays (3) show further significant increases in skin tolerance; at these voltages, under proper conditions, only mild skin reactions are obtained even for high deep-tumor doses delivered through a single portal. It is the purpose of this paper to consider the physical basis for this high skin tolerance and to present an explanation which depends not only on the distribution of ionization produced by such radiation in the region immediately below the skin, but also on the energy of the individual ionizing particles.

## NATURE OF BIOLOGICAL REACTION TO RADIATION

It is well understood that the biological effect of roentgen rays is due to the ionization produced by the absorption of radiation energy in tissue. The nature of this biological reaction is complex; it depends on the amount of ionization produced in the tissue and is affected by the rate at which this total dose is delivered. In the limit, a sufficient amount of ionization results in the immediate demise of the irradiated cell, presumably because of extensive change in the physiochemical structure of the cell protoplasm. At lower dose levels, death of cells may occur because of various kinds of direct cell and neighboring tissue injury; at the threshold dose the complete recovery of the cell is realized. These responses to irradiation vary extensively from one biological material to another

and are dependent on the physical conditions of irradiation.

The effective ionization in all cases is provided by energy removed from the tissue-traversing radiation by photoelectric absorption, Compton scattering, or pair-production. In these interactions of radiation with matter, electrons are accelerated with some or all of the energy of the incident radiation photons. These initial electrons in turn create, by collision with atoms along their path, the many secondary electrons which produce the principal biological effect. These secondary electrons, each possessing an energy generally well in excess of the binding energy of molecules, are capable of exciting and ionizing the atoms with which they in turn collide. The effect of such transfer of energy is often to break up the molecules containing the energized atoms. All manner of molecular disintegrations may occur, from a single break in a long chain molecule to fairly complete reduction to simple atoms. There is abundant evidence (4) that the ease of disruption, or radiosensitivity, is the greater the larger and more complex the molecular structure. Under some circumstances, the effect of energy transfer by electron bombardment may be the development of a more complex molecule, as in the formation of hydrogen peroxide by the irradiation of water.

## ENERGY ABSORBED AND ITS EFFECT IN UNDERLYING TISSUE

Particularly because the ultimate biological reaction is so exceedingly complex, it is fortunate that the total number of ions formed by the radiation per unit volume of tissue can be determined from measure-

<sup>1</sup> From the Department of Electrical Engineering, Massachusetts Institute of Technology, Boston, Mass. Presented at the Thirty-third Annual Meeting of the Radiological Society of North America, Boston, Mass., Nov. 30-Dec. 5, 1947.

ments made with appropriate air ionization chambers. When roentgen radiation traverses a thick section of matter, the absorption of a given amount of its energy produces the same number of ions regardless of the quality of the radiation within wide limits (5). Thus, for example, both 100-kv. roentgen rays and gamma rays produce the same ionization when in each case the same amount of radiant energy is absorbed per gram of the material. Roentgen-ray energy is absorbed at the rate of about 85 ergs per gram of air when a quantity of one roentgen passes through it. About the same energy absorption is realized per gram of water or soft tissue, since these materials have nearly the same atomic number as air.

Low-voltage roentgen rays which undergo photoelectric absorption and exceedingly high-energy roentgen rays which are absorbed by the pair-production mechanism both exhibit marked dependence on the atomic number of the absorber. Roentgen rays of intermediate energy, produced by one to several million volts, are absorbed primarily by the Compton process, which depends directly on the electron density and is nearly independent of atomic number. For such supervoltage radiation, the energy absorption rate of 85 ergs per gram per roentgen holds closely for a wider range of absorber materials. In experiments with high-energy cathode rays (6), it has likewise been confirmed that the absorption of about 85 ergs of electron energy in one gram of air or equivalent material produces ionization corresponding to that produced by one roentgen of radiant energy delivered to one gram of that material.

If energy absorbed were the sole factor, one might believe that a given amount of such energy converted into ionization would have the same biological effect in a given tissue irrespective of the quality of the parent radiation. This is, indeed, approximately confirmed for underlying tissue exposed to radiation such as roentgen rays, gamma rays, or cathode rays. Gall *et al.* (7) showed that the marked difference

between the effect of 200-kv. and 1,000-kv. radiation on the skin of animals disappeared when the skin was moved under the surface by the application of a few millimeters of wax. Several investigators (8) have found that the biological effect of roentgen rays appears to be independent of quality when the indicator is well beneath the surface and the physical conditions of irradiation are similar (9). There is, in fact, much physical reason to believe that the biological effect of widely different qualities of roentgen radiation will be closely the same per unit of energy absorbed in homogeneous tissue provided that this tissue is below the surface, where the beam is fully in equilibrium with its secondaries.

#### SKIN REACTION TO RADIATION OF DIFFERENT QUALITIES

In the region of the skin, however, the biological effect is strongly dependent on the quality of the incident radiation. Of the many biological indicators, the erythema produced on the human skin has perhaps received the most extensive study since the tolerance of the skin is commonly a limiting factor in radiation therapy. The rise of the threshold erythema dose<sup>2</sup> with roentgen-ray voltage is shown in Figure 1, over the range from 100 to 3,000 kv. The lower curve shows the threshold erythema dose for radiation administered in single doses. The values for 2,000-kv. and 3,000-kv. radiation were determined from fractionated treatment data on the upper curve by using ratios obtained by Quimby and MacComb (10).

The high threshold dose for radiation of several million volts energy is particularly noteworthy. Applied in daily doses of 300 r, over 4,000 r of 2,000-kv. radiation and over 5,000 r of 3,000-kv. radiation failed occasionally to elicit any erythema,

<sup>2</sup> Threshold erythema dose is defined, following Failla, as that dose which causes a just perceptible change in the skin of about 80 per cent of the subjects and no discernible discoloration in the remainder in two to four weeks after exposure. This biological unit is recognized as approximate and subject to individual variations, but still reasonably accurate for clinical use.

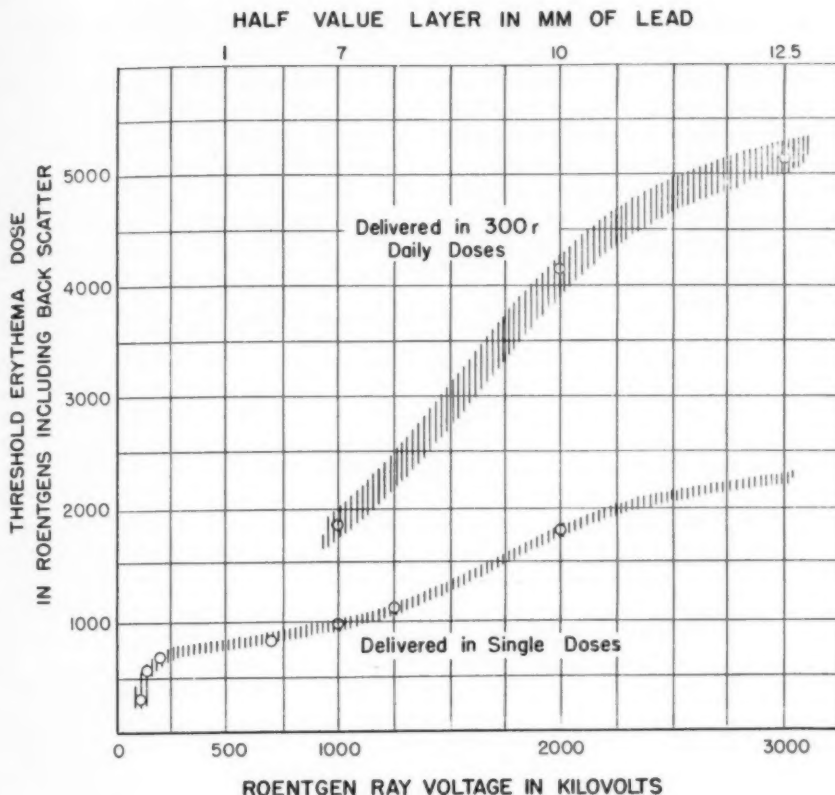


Fig. 1. Threshold erythema dose in roentgens for multiple and single dose therapy at different voltages.

and only faint reactions were discernible in the other cases. These observations were made by Dresser (3), mostly on abdominal skin, with a  $10 \times 10$ -cm. field, a target-skin distance of 100 cm., and an equivalent filtration of 9 mm. of lead. The doses were measured in the region of highest ionization density (corresponding to the peaks of Figure 2) and therefore include all scatter. The high skin tolerance is realized only with uncovered skin and at the portal of entry. It is clear that for 2,000- and 3,000-kv. radiation, under proper conditions, the tolerance of the skin is such that adverse skin reactions are unlikely even for the highest deep tumor doses that may in practice be directed through a single portal. These threshold values are substantially higher than the observed value for gamma rays, which approximate 2,000-kv. roent-

gen rays in quality. Gamma rays are often accompanied by many secondary electrons from the source or from the filter placed in the beam to stop these secondaries.

#### DISTRIBUTION OF ION DENSITY IN THE REGION OF THE SKIN

The explanation for the higher threshold erythema dose of high-energy radiation is usually based on the observation that the ionization is not maximum at the surface, but at a distance under the surface equal to the mean range of the initial electrons (11, 12). Study of the ionization density at successive levels below the skin shows that this important effect accounts only in part for the increase in threshold erythema dose. Figure 2 shows the measured distribution of ionization in the region immediately below the skin for filtered roentgen rays pro-

duced by constant-potential electrons with 200, 1,000, 2,000, 3,000, and 4,000 kv. of energy. With 1,000-kv. radiation, the intensity at the extreme skin surface is 60 per cent of the maximum and has increased to about 95 per cent of the maximum at a depth of 1.0 mm.; for 2,000-kv. radiation the surface intensity is 40 per cent and the 95 per cent value is over 2.0 mm. below;

#### QUALITY EFFECTS IN SKIN

Analysis of Figures 1 and 2 shows that the lowered ionization density at the skin, though considerable at supervoltages, is not sufficient of itself to account for the entire increase in skin tolerance. Moreover, no such lowered surface ionization is obtained with radiation of less than 500 kv. energy. An additional and presumably a quality

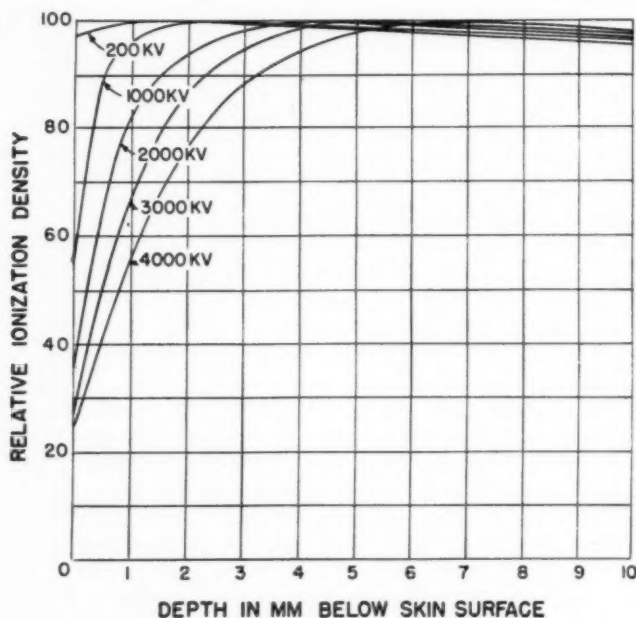


Fig. 2. Distribution of ionization in the first 10 mm. below the skin for 200-kv. and for supervoltage roentgen rays filtered as indicated below, with a  $10 \times 10$ -cm. field and 100 cm. distance.

Kilovoltage	Filtration
200	0.5 mm. Cu
1000	2.0 mm. Pb + 5.0 mm. Cu
2000	6.0 mm. Pb + 5.0 mm. Cu
3000	10.0 mm. Pb + 5.0 mm. Cu
4000	20.0 mm. Pb + 5.0 mm. Cu

for 4,000-kv. radiation the surface dose is 25 per cent of the maximum, which now occurs over 4.0 mm. below the skin. Evidently with supervoltage rays, because of the increased range of the forward-directed electrons produced by the entering roentgen rays, the skin region receives only a fraction of the maximum ionization dose, and this fraction becomes smaller the higher the voltage.

effect of the radiation must therefore be involved. The explanation proposed here is that in the region of the skin the ionization contains electrons of higher average energy than are found farther below the surface and that such higher-energy electrons are in themselves less effective biologically.

That a larger percentage of higher-energy electrons are present in the first

few millimeters of tissue follows from a consideration of the x-ray absorption process. With supervoltage x-rays the absorption of radiation energy, largely by the Compton process, results in forward-directed electrons which may have energies up to very nearly the energy of the absorbed photon. Beginning at the surface, each elementary thickness contributes secondary electrons which continue on into the tissue below. A two-million-volt electron produced in this way, for example, will proceed a total distance of 1.0 cm. in tissue in a generally forward direction and during this travel will lose energy by collision with the electrons of the atoms of the cells through which it progresses. The ion density produced by such an electron will be higher near the end of its path than in the region where it is traveling with considerable energy. Viewed statistically, this process produces ionization containing a larger percentage of high-energy electrons in the region nearest the surface. As one progresses below the surface, the average energy of the electrons reduces to a lower value, which becomes nearly constant after the initial high-energy electrons are in equilibrium with their more numerous low-energy descendants. This condition of electronic equilibrium is reached at that depth below the surface which corresponds to the forward range of the electrons of highest energy. Thus, as the radiation progresses through the first few millimeters below the surface, there occur both an increase in the number of charged particles per unit volume and a reduction in their average quality or energy content.

While the existence at the entering surface of electron ionization of higher energy can be most simply described in the case of supervoltage roentgen rays, a similar effect is realized with roentgen rays in the energy range from below 50 to over 200 kv. The soft 50-kv. radiation establishes secondary photon and electron equilibrium very close to the surface, while 200-kv. radiation, for example, requires a correspondingly greater distance. This does not significantly affect the location of the region of maximum ion

density, which remains on or close to the surface, but does affect the average energy of the ionizing electrons found in this region. The marked increase in skin tolerance observed at the lower roentgen-ray voltages may be explained by these considerations.

Electrons of relatively high energy may be expected to be less effective per micron of path in inducing photochemical and biological reactions than electrons which possess only tens of volts of energy. Depending on its nature, excitation of an individual atom may require only a fraction of a volt, and complete ionization can be effected by a few volts of energy. The structure of complex molecules can be affected by the small amounts of ionizing energy corresponding to that required for the ionization of single atoms. The study of ionization processes in gases has shown that the probability of excitation and ionization of atoms begins to diminish when the impinging electron has more than several times the minimum amount for that reaction. Accordingly, one would expect that the photochemical and biological effectiveness of electrons would be a maximum for energies from a fraction to a few volts and would be relatively less for electrons with energies of hundreds of volts and higher. The higher bactericidal effectiveness of 2,500-ångström ultraviolet radiation, for example, is accomplished by photons which possess a maximum of about 5 electron volts of energy.

Explanation of the relative biological effectiveness of radiation has also been based on the density of ionization produced along the tracks of the individual ionizing particles (6). This point of view argues that maximum biological effect is realized when the ion density per micron of path is sufficient to produce an adequate number of ions in the irradiated cell volume and that the biological efficiency diminishes when a great excess of ions is produced in each cell. The low linear ion density of gamma rays and supervoltage x-rays is appropriate for the efficient inactivation of small units such as viruses, which seem to

require only single ionization events. For the chromosome structural changes which are an important part of the response of normal and malignant tissue, it has been shown that higher ion densities corresponding to lower-energy radiations are more effective. The linear ion density theory thus supports qualitatively the expectation that the biological effectiveness of supervoltage roentgen radiation would increase from an initial value at the entering surface to a substantially constant and higher value at that distance below the surface where the secondary electrons have attained energy equilibrium at a lower average value.

#### SUMMARY

With supervoltage roentgen rays the ionization produced in the surface region is both less than that available some distance below and composed of electrons of relatively high average energy and therefore lower biological effectiveness. These considerations make possible an adequate physical explanation of the increased skin tolerance of high-energy radiation. This effect must disappear almost completely when the radiation beam has traversed a distance below the surface equal to the range of the highest-energy electrons which the beam can produce. Beyond this distance the electromagnetic radiation is always accompanied by the full complement of low-energy secondaries so that the average energy of the electron ionization is little different from that produced by low-voltage roentgen rays. Thus, for tissue lying deeper than the level of maximum ionization density, the biological effect per erg of energy absorbed should be closely the same throughout the roentgen-ray spectrum.<sup>3</sup>

<sup>3</sup> Clinical difference between various qualities of radiation may be observed in the depth of an inhomogeneous absorber because of differences in the physical distribution of ionization. In addition to the different penetrabilities of low- and high-energy radiation, the atomic number of the absorber may influence the distribution. The absorption of supervoltage radiation depends on the density and is nearly independent of the atomic number of the absorbing atoms. Low-energy radiation is strongly dependent on the atomic number because of the photoelectric absorption process. Such softer radiation would thus tend to affect selectively the bony tissue because of its higher atomic number as well as specific regions within individual cells.

Only at the entering skin should the biological effect become progressively less the higher the voltage.

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#### REFERENCES

1. MEYER, W. H., AND GLASSER, O.: Further Studies on Influence of Radiation Quality on the Erythema Dose Measured in Physical Units. *Radiology* 8: 311-316, 1927.
2. DRESSER, R., RUDE, J. C., AND COSMAN, B. J.: Differences Between 200 Kilovolt and Supervoltage Roentgen Therapy. *Radiology* 34: 12-16, 1940.
3. DRESSER, R.: Further Observations on the Use of Three Million Volt Roentgen Ray Therapy. *Radiology* 50: 645-648, 1948.
4. LEA, D. E.: Actions of Radiations on Living Cells. New York, Macmillan Company, 1947.
5. GRAY, L. H.: Absolute Measurement of Gamma-Ray Energy. *Proc. Roy. Soc., London, ser. A* 156: 578-596, 1936.
6. TRUMP, J. G., VAN DE GRAAFF, R. J., AND CLOUD, R. W.: Cathode Rays for Radiation Therapy. *Am. J. Roentgenol.* 43: 728-734, 1940.
7. GALL, E. A., LINGLEY, J. R., AND HILCKEN, J. A.: Comparative Experimental Studies of 200 Kilovolt and 1000 Kilovolt Roentgen Rays. The Biologic Effect on the Skin of the Albino Rat. *Am. J. Path.* 17: 319-334, 1941.
8. GRIMMETT, L. G.: Personal communication, 1946.
9. FAILLA, G., AND MARINELLI, L. D.: Measurement of the Ionization Produced in Air by Gamma Rays. *Am. J. Roentgenol.* 38: 312-343, 1937.
10. MACCOMB, W. S., AND QUIMBY, E. H.: Rate of Recovery of Human Skin from the Effects of Hard or Soft Roentgen Rays or Gamma Rays. *Radiology* 27: 196-207, 1936.
11. MAYNEORD, W. V.: Energy Absorption. *Brit. J. Radiol.* 13: 235-247, 1940.
12. TRUMP, J. G., AND CLOUD, R. W.: Production and Characteristics of 3000 Kilovolt Roentgen Rays. *Am. J. Roentgenol.* 49: 531-535, 1943.
13. GRAY, L. H.: Comparative Studies of the Biological Effects of X Rays, Neutrons and Other Ionizing Radiations. *Brit. M. Bull.* 4: 11-18, 1947.

#### DISCUSSION<sup>1</sup>

**Milton Friedman, M.D.** (New York, N. Y.): I would often rather have 200-kv. x-rays using multiple-port or rotation technic than I would three-million-volt x-rays using one single portal.

<sup>1</sup> This Discussion pertaining to the papers by Dr. Dresser and Dr. Trump is part of the discussion of a larger group of papers on therapy in which these were included. The remainder of the group, along with the rest of the discussion, will appear later in *RADIOLOGY*.

I don't mean to decry the single portal with million-volt x-rays, but the amount of damage to adjacent normal tissues will be significantly diminished by multiple-small-portal technic. Supervoltage irradiation through a single port has a few specific indications; it is particularly effective when irradiating massive tumors, such as massive retroperitoneal sarcoma, wherein the beam of radiation traverses nothing but skin, tumor, and skin. The vulnerable intestines have been pushed aside by the tumor. When supervoltage irradiation plus rotation or multiple-portal technic is employed, then the physical distribution of radiation described by Dr. Trump in a recent paper (in which he demonstrated with x-ray films that you can build up a "hot spot" 4 or 5 cm. in diameter in the midst of a chest with little or no radiation) approximates interstitial irradiation.

**R. R. Newell, M.D.** (San Francisco, Calif.): In regard to the multiple-port technic, it is quite easy to show by physics and geometry what an enormous advantage it has for deep-seated lesions. The secret of success, of course, is an infinite degree of precision. Yet, to get the ultimate from multiple-port technic the precision does have to approach perfection.

In regard to Dr. Trump's remarks about the dosage at the surface, I have long been interested in this problem. There are two things I would like to bring out. First, I think it is easy to overestimate the effect of the columnar density of ionization along the track of high-speed electrons, for this reason: the difference in columnar density between the beginning part of the high-speed electrons in the first millimeter or two of the skin, before they have come into equilibrium with their primary, is small compared to the columnar density toward the end of the path. It might be a ratio of 1 to 10 on the average. Yet, we see in comparing x-ray treatment with neutron-ray treatment, where we are dealing with the difference in columnar density of 100 times, we still have reached only a biologic factor of from 2 to 5. So that one cannot expect an overwhelming difference in the biologic factor between the first part of the path of the high-speed electron and the

end of it. It isn't as though you had a single high-speed electron going through the skin and had the very low specific ionization at the first part of the path, at the surface, and very high specific ionization in the last part, at a centimeter depth; it can't be as pure as that because the composition is always very mixed. I would be inclined to give a good deal more weight to Dr. Trump's first factor, which is the delay in coming into equilibrium with the secondaries, it being the secondaries which are actually doing the ionization.

The second thing has to do with the injury to the skin. The tolerance of the skin is badly injured by the fact that, when a large enough dose is used to knock out the epithelium, then one gets a superficial infection, so that the skin might be able to put up with the large dose if it were not for the fact that there is inevitably added an infection on top of it. It is astonishing what a very large tissue dose can be borne by tissues close under the skin when those doses are applied in the operating room with the skin turned back and pulled over afterward.

**Richard Dresser, M.D.** (closing): I would like to say a word or two about cross-fire technic. Undoubtedly we can do a good job with cross-fire radiation at 200 kv., but it takes a great deal of care and precision, as has been pointed out, and I tried to stress the fact that with supervoltage roentgen treatment the technic is far simpler; one can select a portal and mark it off if one likes and the technician can go ahead with the routine exposures. I feel very strongly that this is a definite step in advance.

**John G. Trump, Sc.D.** (closing): I appreciate very much the comments of Dr. Friedman and Dr. Newell. I find myself mostly in agreement with what Dr. Newell suggested. It is our impression that most of the increased skin tolerance with supervoltages is related to the progressive movement of the peak dose to a region further below the radiosensitive skin. The residual, hitherto unexplained tolerance may be about 20 per cent. This may be accounted for by the observation that the average energy of the electron ionization diminishes as the beam moves under the skin.

#### SUMARIO

##### Base Física para la Alta Tolerancia de la Piel hacia los Rayos X de Supervoltaje

Con los rayos X de supervoltaje la ionización producida en la región superficial es menor que la obtenible a alguna distancia más adentro a la vez que se halla compuesta de electrones de energía media relativamente alta, y por consiguiente, de menor efectividad biológica. Estas con-

sideraciones permiten ofrecer una explicación física adecuada de la hipertolerancia cutánea a la radiación de alta energía. Este efecto tiene que desaparecer casi por completo cuando el haz de rayos ha atravesado por debajo de la superficie una distancia igual al alcance de los electrones de

energía máxima que puede producir el haz. Más allá de dicha distancia la irradiación electromagnética va siempre acompañada del pleno complemento de rayos secundarios de baja energía, de modo que la energía media de la ionización electrónica varía poco de la producida por rayos X de bajo voltaje. Por esa razón, para los

tejidos más profundos que el nivel de la densidad máxima de ionización, el efecto biológico por erg de energía absorbida debe ser bastante idéntico en todo el espectro de rayos X. Tan sólo en la piel de entrada debe dicho efecto volverse progresivamente menor a mayor voltaje.



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# Venous Calcification in Banti's Syndrome

## Report of a Case<sup>1</sup>

ALAN R. BLEICH, M.D.,<sup>2</sup> and CHARLES S. KIPEN, M.D.<sup>3</sup>

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VENOUS CALCIFICATION within the abdominal cavity, with the exception of phlebolith formation, is an extremely rare condition. Roentgen evidence of its occurrence is not to be found in the literature, although arterial changes have been frequently described. The few reported instances of venous calcification do not include radiographic studies, the changes having been found at autopsy or surgery. The occurrence of venous sclerosis in Banti's syndrome is especially interesting in view of the evidence that portal bed obstruction is probably the etiologic agent. Because of these features and the definite and striking roentgen picture, the following case was thought to be worthy of presentation.

### CASE HISTORY

L. L. B., a 49-year-old white male, was admitted to the Medical Service of the Wadsworth General Hospital, July 14, 1946. From 1919 to 1929, he had severe and repeated gastric hemorrhages, for which he was frequently hospitalized. He had been told that he had an enlarged spleen. In 1929, at another hospital, a splenectomy was done. Records obtained from that hospital are essentially unrevealing. A clinical diagnosis of Banti's or Gaucher's disease was made but there was no pathological examination. Following splenectomy, the patient remained well until May 1946, when he had a severe gastrointestinal hemorrhage, vomiting bright red blood and passing tarry stools. This occurred while he was on his honeymoon, and he was taken to a local hospital, where he had several more severe hemorrhages. He was later treated with multiple transfusions and was told he had a bleeding duodenal ulcer, shown radiographically. Three weeks later, while en route to the West Coast, he had another severe hemorrhage and one week before admission he had still another. He was hospitalized twice, en route, because of recurrent hemorrhage.

On admission, the patient appeared rather thin and very pale. The physical examination was essentially negative except for a left upper rectus scar, a liver which was palpable two finger-breadths below the costal margin on deep inspiration, and internal hemorrhoids. Occult blood was found in the stool. The erythrocyte count was 3,100,000 with 56 per cent hemoglobin. The platelet count, prothrombin time, and fragility tests were normal. The urine examination revealed three-plus albumin, numerous red and white blood cells, and a low specific gravity. The urea nitrogen was 31.8 mg. per cent.

The patient had several hemorrhages while on the Medical Service, the erythrocyte count at one time falling to 1,300,000 with 26 per cent hemoglobin. He was treated with multiple transfusions and was seen in surgical consultation. It was felt that his condition was too poor for any major surgical procedure at this time and conservative therapy and repeated transfusions were therefore recommended. When he improved he was transferred to the Surgical Service.

Very shortly after the patient's arrival on the Surgical Service, severe hemorrhages again occurred and repeated transfusions were instituted. The urea nitrogen rose to over 50 mg. per cent and the urine showed a low specific gravity with numerous red and white blood cells. X-ray studies showed one constant finding, a density running transversely across the upper mid-abdomen and lying behind and below the stomach in the region of the pancreas. Several gastro-intestinal examinations were done. These were interpreted by different observers as indicating possible perforating duodenal ulcer with outpouching into the pancreas, calcification of the pancreas, or calcification of the portal vein. However, x-rays also showed bilateral ureteral calculi, and in view of the elevated urea nitrogen and low urinary specific gravity, it was decided that the patient would be a poor surgical risk. Accordingly, as soon as his general condition improved, he was transferred to the Genito-Urinary Service, where he had two separate operations for the removal of ureteral calculi. On Sept. 25, 1946, a right pyelolithotomy was done, and on Oct. 8, a left pyelolithotomy. Recovery was satisfactory; the urea nitrogen dropped to within normal limits and phenolsulfonphthalein

<sup>1</sup> Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors. Accepted for publication in August 1947.

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Fig. 1. The calcified vein is seen at the level of the 12th dorsal vertebra. It measures 10 X 2 cm. Other findings include a right hydroureter and a left ureteral calculus.

function tests showed 57 per cent of total function. The erythrocyte count rose to 3,000,000 with a 65 per cent hemoglobin, and the patient was transferred back to the Surgical Service on Nov. 4, 1946.

Following re-evaluation of the findings, it was felt that the patient probably had a calcified portal and/or splenic vein causing portal hypertension with bleeding secondary to gastric or esophageal varices. An esophagoscopy was done Nov. 22, 1946, which failed to reveal esophageal varices and following which another moderate hemorrhage occurred.

On Dec. 4, 1946, exploratory laparotomy was done under general anesthesia. Dense adhesions were found between the anterior parietal peritoneum and the greater omentum, transverse mesocolon, and stomach. These adhesions contained numerous dilated veins and formed what was felt to be an adequate natural omentopexy. Some enlarged veins could be seen in the wall of the stomach, but no definite gastric or esophageal varices were noted. There was no evidence of ulcer. The long stump of the splenic vein and the portal vein posteriorly to the descending portion of the duodenum were found to be calcified, accounting for the unusual radiographic picture, and the liver presented grossly the picture of portal cirrhosis. It was felt that the patient had a late Banti's syndrome with portal hypertension



Fig. 2. Spot films reveal considerable tortuosity of the calcified vein and show it extending across the mid-line.

secondary to occlusion of the splenic and portal veins by calcification. The abdomen was closed with no attempt at any therapeutic procedure. Spleno-renal and post-caval anastomosis were considered but were not deemed feasible because of the calcification of the veins involved, the poor status of the kidneys, and the poor general condition of the patient.

The postoperative course was entirely uneventful. No hemorrhages occurred and the blood count rose and was maintained at a level of 75 per cent hemoglobin and an erythrocyte count of 4,250,000; the leukocyte count remained at about 7,000 with a normal differential count. The urine continued to show the presence of albumin and pus. The patient was allowed to leave the hospital on several occasions, without untoward effects, and was discharged on Jan. 15, 1947.

#### COMMENT

In 1883 Banti described the syndrome which bears his name. According to his original concept the etiologic agent was a toxic substance which first caused splenomegaly, with hepatic and other sequelae. Banti described three stages, which he said were specific for this disease, and noted that there were pathognomonic lesions in the spleen. With respect to venous calcification, Trevor (1) quoted Banti as follows: "In the splenic vein one sees the lesions of a sclerotic endophlebitis, . . . and true atheromatous and calcareous patches may be found. Such changes may spread into the portal vein." Trevor's case, that of a male who died from repeated alimentary tract hemorrhages, came to autopsy. Marked calcification of the splenic artery, splenic vein, inferior mesenteric vein, left gastro-epiploic vein, superior mesenteric

and portal vein up to the point of division were shown. The liver was greatly enlarged and cirrhotic.

Osler (2) described a case of fibrous obliteration of the portal vein, with calcification of the gastric, splenic, and mesenteric veins. Trimble and Hill (3) report a case of congestive splenomegaly due to portal stenosis without hepatic cirrhosis, in which rings of calcification were seen in the portal vein in addition to calcification of aneurysms of the splenic artery.

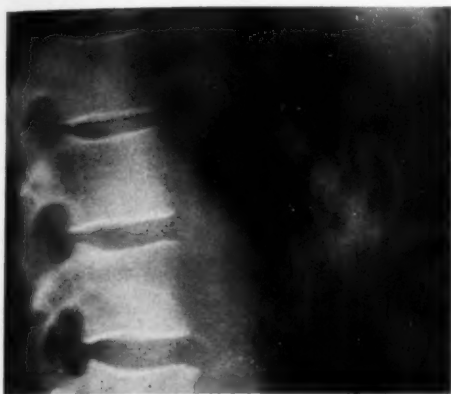


Fig. 3. A lateral view shows the calcified vein in cross section.

Christian (4), in stating the present concept of Banti's syndrome, says that the etiology lies in splenic vein obstruction. He notes that the splenic as well as the portal vein may be enormously dilated and show atheromata and calcification. Ravenna (5), however, lists twelve cases of known splenic vein obstruction in none of which was there any degree of splenomegaly. He believes that obstruction to the splenic venous channel will cause atrophy of the spleen.

Whipple (6) states definitely that the modern concept of Banti's syndrome does not conform to the specific pattern laid down by Banti and that it is not a disease entity; hence the use of the term syndrome. He believes that the syndrome is characterized by a secondary anemia, leukopenia, thrombocytopenia, and splenomegaly, with repeated severe gastro-intestinal hemor-



Fig. 4. The juxtaposition of calcification and the barium-filled stomach is well shown.

rhages. The liver may or may not show cirrhosis. Esophageal varices may be a feature. The condition is now believed to be due to mechanical obstruction to the flow of blood within the portal bed. This obstruction may be intra- or extrahepatic. The Spleen Clinic at Presbyterian Hospital, New York City, had 6 cases of sclerosis of the portal vein out of 174 cases diagnosed as Banti's syndrome. Bachman (7) described eleven cases of arteriosclerosis of the splenic artery, in none of which were there similar changes involving the splenic vein.

#### CONCLUSION

A case of Banti's syndrome is presented, with roentgen evidence of calcification of the splenic and portal veins confirmed at operation.

A review of the pertinent literature fails to show any account of similar roentgen findings.

NOTE: This case is presented with the kind permission of Dr. C. G. Lyons, Chief of the Radiological Service, and Dr. Charles H. McIntyre, Chief of the Surgical Service, General Hospital, V. A. Center, Los Angeles, California.

The authors wish to express their thanks to Mr. W. L. M. Martinson for his reproductions of the original radiographs.

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## REFERENCES

1. TREVOR, R. S.: Multiple Aneurysms of Splenic Artery Associated with Calcification and Dilatation of the Portal Vein and Its Radicles. *Tr. Path. Soc., London* **54**: 302-308, 1903.
2. OSLER, WILLIAM: Principles and Practice of Medicine. New York, D. Appleton & Co., 4th Ed., 1901, p. 774.
3. TRIMBLE, W. K., AND HILL, J. H.: Congestive Splenomegaly (Banti's Disease) Due to Portal Stenosis Without Hepatic Cirrhosis; Aneurysms of the Splenic Artery. *Arch. Path.* **34**: 423-430, 1942.
4. CHRISTIAN, HENRY A.: *Osler's Principles and Practice of Medicine*. New York, D. Appleton-Century Co., 14th Ed., 1942, p. 984.
5. RAVENNA, PAOLO. Splenoportal Venous Obstruction Without Splenomegaly; Further Contribution to Pathogenesis of Fibrocongestive Splenomegaly (Banti's Syndrome). *Arch. Int. Med.* **72**: 786-794, 1943.
6. WHIPPLE, ALLEN O.: Problem of Portal Hypertension in Relation to the Hepatosplenopathies. *Ann. Surg.* **122**: 449-475, 1945.
7. BACHMAN, ARNOLD L.: Calcifications in the Splenic Region. *Am. J. Roentgenol.* **41**: 931-949, 1939.

## SUMARIO

## Calcificación Venosa en el Síndrome de Banti

Preséntase un caso de síndrome de Banti, con signos roentgenológicos de calcificación de las venas esplénica y porta. Un repaso de la literatura pertinente no ha revelado hallazgos roentgenológicos semejantes.



# Malignant Plasmocytoma of the Nasopharynx

## A Case of Multiple Myeloma Primary in the Nasopharynx<sup>1</sup>

CAPT. MAURICE M. GREENFIELD, M.C., A.U.S.

IN 1935 MATTICK and Thibaudeau (1), reporting a case of multiple plasma-cell tumors in the upper respiratory tract, stated that "the occurrence of plasma-cell tumors of extramedullary origin is of sufficient interest and rarity to merit the report of even a single case." Others have repeatedly noted the infrequency of this lesion and expressed similar opinions. Haines (2) suggested that further reports were required of "metastasizing plasma-cell tumors of the upper respiratory tract," and recognized three clinical types, based on the presence or absence of lymph node and skeletal involvement.

Of further interest is the fact that a review of the literature reveals the existence of considerable confusion concerning the malignant character of this group of lesions. Frank (3) referred to the indiscriminate use of the term, "plasmocytoma" for both inflammatory and neoplastic (benign and malignant) accumulations of plasma cells.

Hellwig (4), following a thorough review of the literature, classified the extramedullary lesions into five groups, as follows: (1) simple benign tumors; (2) multiple benign tumors; (3) malignant tumors without metastasis; (4) malignant tumors with lymph node metastasis; (5) malignant tumors with osseous metastasis. In his collected series of 128 cases, there were 9 originating in the air passages and metastasizing to bones; 4 of these showed, in addition, metastasis to lymph nodes. All cases terminated fatally. Hellwig also noted the futility of predicting the clinical course or prognosis from the histologic appearance of the lesion.

Still others have taken exception to the view that extramedullary plasmocytomas occur as benign lesions. Figi, Broders,

and Havens (5), reporting 11 cases of plasma-cell tumors of the respiratory tract found no evidence to support the conception that the lesion is benign. They felt that, while it is true that extramedullary plasmocytomas may vary greatly as to their malignancy and course, there appears to be little evidence to indicate that they are not all malignant lesions, and they should be so treated.

The case which is the subject of this report is interesting because of its occurrence in an 18-year-old male, and because it adds further evidence to the view that these lesions should be regarded as malignant neoplasms.

### CASE REPORT

A 20-year-old white soldier was said to have been well until January 1945, when he noticed a swelling on the right side of the neck. This had appeared suddenly, remained well localized, and grew slowly over a period of several months. It was unassociated with pain, fever, or weight loss. Previous medical history was said to be non-contributory. On July 12, 1945, the patient was inducted into military service and was promptly hospitalized because of the discovery of a mass of enlarged right cervical nodes.

Physical examination showed the patient to be well developed and well nourished. He did not appear acutely or chronically ill. His temperature was 98.6° F., pulse 84 per minute, respirations 24 per minute, blood pressure 125/80 mm. The mass of nodes on the right side of the neck lay deep to the belly of the sternomastoid muscle and measured 10.0 × 7.5 × 2.5 cm. It was firm, moderately fixed, and non-tender. Other physical findings were not significant.

The peripheral blood contained 4,100,000 red blood cells per cubic mm. and 9,500 white blood cells, with 58 per cent neutrophils and 40 per cent lymphocytes. Urinalysis was negative, as was the Kahn serologic test. Blood chemistry findings, including total serum proteins, blood urea nitrogen, and blood sugar, were normal. The tuberculin skin test was negative.

The patient was afebrile and asymptomatic, ex-

<sup>1</sup> From the Radiation Therapy Section, Walter Reed General Hospital, Washington, D. C. Accepted for publication in August 1947.

cept for the localized lymphadenopathy. On July 30, 1945, the enlarged nodes in the right side of the neck were removed. They were reddish brown in color, appearing well encapsulated and firm. The postoperative course was uneventful.

Sections prepared from the mass of nodes were reported as "malignant lymphoma, reticulum-cell sarcoma type" (Fig. 1), and the patient was transferred to Walter Reed General Hospital for radiation therapy on Aug. 22, 1945.

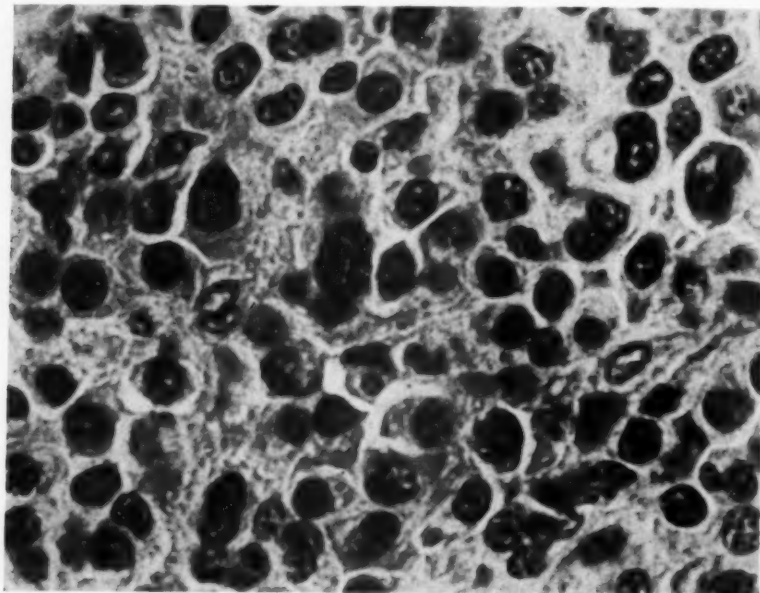


Fig. 1. Biopsy of lymph node ( $\times 965$ ), showing replacement by cells with eccentric nuclei. The nuclear material is loose and shows the cartwheel arrangement of plasma cells.

Examination on admission was not remarkable. There was a healed biopsy scar on the right side, overlying the anterior cervical triangle at the level of the upper portion of the thyroid cartilage. Numerous small, pea-sized nodes were palpable in the anterior and posterior cervical chain bilaterally. There was no evidence of enlarged axillary or inguinal nodes, or of abnormal abdominal masses. Radiographic study of the chest was reported as negative.

Because of the histologic diagnosis of reticulum-cell sarcoma, roentgen therapy was administered to the cervical chain of nodes bilaterally. From Sept. 13 to Oct. 11, 1945, high-voltage irradiation was given for a total dose of 4,000 r (measured in air, with backscatter) to the nodes on each side of the neck. The physical factors were as follows: 200 kv., 20 ma., 50 cm. target-skin distance, 0.5 mm. Cu and 1.0 mm. Al filtration, half-value layer 1.0 mm. Cu, 38 r per minute (in air).

The enlarged nodes shrank rather slowly, suggesting that either the diagnosis of reticulum-cell sar-

coma was erroneous, or that this was an extremely radioresistant reticulum-cell lesion. With this thought in mind, the original biopsy of the lymph nodes was reviewed by our Department of Pathology and a diagnosis of plasmacytoma was made. Following revision of the initial diagnosis, a complete skeletal survey and further laboratory studies were done.

Radiographic studies of all bones revealed two separate destructive lesions, involving the left 6th rib

at its vertebral articulation and the right 6th rib in the posterior axillary line (Fig. 2). These lesions were reported as being "consistent with multiple myeloma."

Urinalysis on two occasions during October and November 1945 was positive for Bence-Jones protein. Blood calcium was 12.8 mg. per 100 c.c.; blood phosphorus, 4.9 mg. per 100 c.c.; alkaline phosphatase, 2.22 Bodansky units (normal, 2-9). Total serum proteins were 7.9 gm. per cent; albumin, 4.9 gm. per cent; globulin, 3 gm. per cent; albumin-globulin ratio, 1.63 to 1. The red blood count was 5,150,000, with 90 per cent hemoglobin; the white blood count was 9,700 per cubic millimeter, with 58 per cent neutrophils, 37 per cent lymphocytes, and 4 per cent smear monocytes. No abnormal cells were seen on smear of the peripheral blood.

Upon further questioning, it became apparent that the osseous rib lesions were the source of severe pain in the mid-dorsal spine and right lateral chest. Palliative roentgen therapy was given to these le-

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sions from Oct. 31 to Dec. 1, 1945. A dose of 4,000 r (measured in air with back-scatter) was given to the lesion in the left posterior 6th rib, and 2,600 r (in air) to the lesion in the right lateral 6th rib. There was striking relief of pain, which disappeared on completion of radiation therapy.

The patient felt well for several months and was asymptomatic. His weight remained at 165 pounds, and his appetite was good. He was again seen in June 1946, at which time he complained of severe headaches in the region of the right orbit, numbness of the right half of the upper lip, and right nasal obstruction.

Careful interrogation with respect to the past history, never adequately explored, revealed exceedingly interesting information. In July 1943, three years previously, the patient was hospitalized, while still a civilian, at a private hospital in Port Jervis, New York, because of nasal obstruction. "Multiple polypoid growths" were removed from the nasopharynx, and postoperative irradiation was advised. For some reason, not entirely clear, he failed, however, to receive the x-ray treatments. Additional investigation showed that the tissue originally removed from the nose was diagnosed "plasmocytoma of the nasopharynx."<sup>2</sup>

In view of this pertinent history, nasopharyngoscopic examination was done, revealing a mass in the right nasopharynx, about the size of a large cherry, involving the posterior wall and roof. Radiographic studies of the nasal accessory sinuses and skull were negative.

On Aug. 7, 1946, a biopsy (Fig. 3) of this recurrent, nasopharyngeal tumor was taken under general anesthesia. The tumor bled rather profusely on incision, but bleeding was controlled by local tannic acid application.

The pathologic report was as follows: "One surface of the nodule is partially covered by attenuated, stratified squamous epithelium; lying beneath and adjacent to this are broad sheets of densely packed cells, which, for the most part, are oval to round, having abundant, finely granular, eosinophilic cytoplasm and deeply basophilic nuclei exhibiting prominent chromatin blocking of cartwheel type. Nuclei are for the most part eccentric and show occasional mitoses. In general, the cells are fairly uniform, but scattered giant-cell and multinucleated forms are seen. The stroma is rather sparse but forms trabeculations throughout the tumor mass. Vascularity is prominent. Diagnosis: Plasmocytoma, right nasopharynx."

Following histologic confirmation of our clinical impression, intensive million-volt x-ray therapy was given to the recurrent neoplasm in the nasopharynx. From Aug. 10 to Aug. 31, 1946, a dose of 3,000 r (measured in air, with back-scatter) was delivered to each of five portals, angulated in such fashion as



Fig. 2. Cystic destructive lesions involving the posterior left sixth rib at its vertebral articulation. The right sixth rib shows a pathologic fracture with some reactive bony proliferation.

to cross-fire the bulk of the tumor. This delivered a calculated tumor dose of 6,610 r to the lesion. The physical factors were: 1,000 kv., 3 ma., 70 cm. target-skin distance, 3 mm. tungsten filter, half-value layer 3.6 mm. Pb, 88 r per minute (in air).

Shrinkage of the tumor was slow, considering the moderately large dose which it received, but the complaints of nasal obstruction and orbital headaches disappeared. Because of the residual tumor after six weeks of observation, a post-irradiation biopsy was taken. On Oct. 25, 1946, almost eight weeks following the last x-ray treatment, a biopsy (Fig. 4) was done. This showed viable, residual plasmocytoma, with only slight radiation changes.

Subsequently there developed additional osseous involvement of the floor and medial wall of the right orbit, with unilateral exophthalmos as a result of direct extension from the nasopharynx, as well as distant destructive bone lesions. These were treated palliatively with x-ray therapy, with symptomatic improvement.

When last seen, in July 1947, four years following the original diagnosis, the patient was moderately well in spite of the presence of multiple bone involvement in the skull, ribs, and pelvis, and residual tumor in the right nasopharynx.

#### DISCUSSION

Extramedullary plasma-cell tumors, while rare, form an interesting group, whose precise malignant position has never been clearly defined in the gamut of neoplastic disease. In certain respects they behave not unlike many tumors of the lymphoma group. Probably in rare instances they are misdiagnosed and called benign on the basis of their histologic appearance, confirmed by a short follow-up period, without recurrence. In the latter

<sup>2</sup> Slide No. 237687, courtesy Institute for the Study of Malignant Diseases, Buffalo, N. Y.

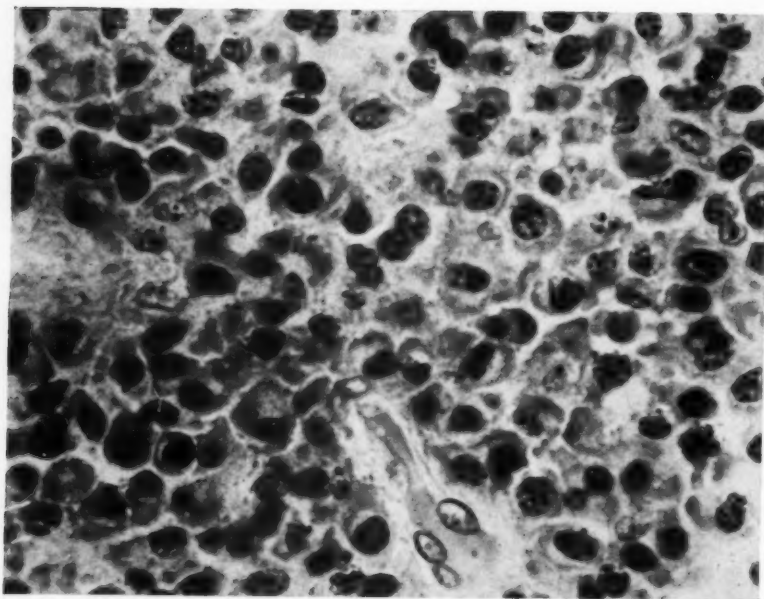


Fig. 3. Section ( $\times 705$ ) of the tumor in the nasopharynx. The tumor consists almost entirely of cells with nuclei varying in size and eccentrically placed. The striking feature is the characteristic plasma cell with its nuclear chromatin arranged in cartwheel fashion.

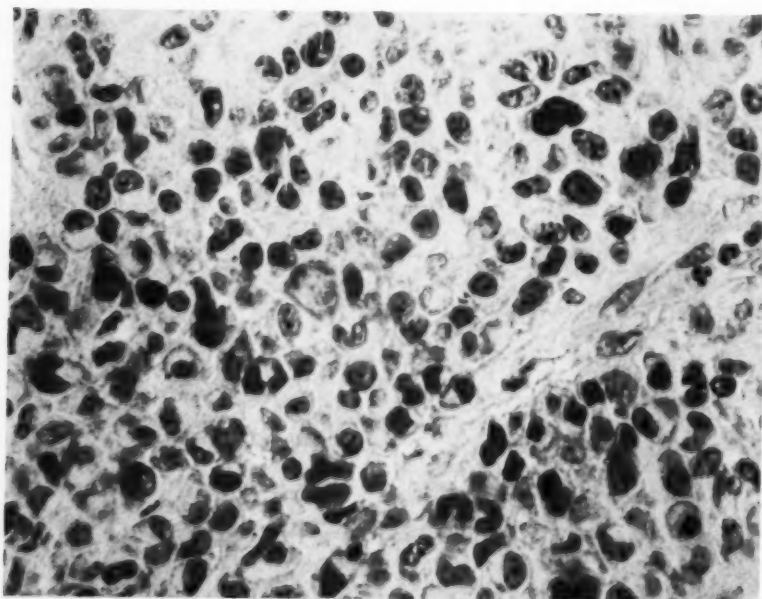


Fig. 4. Post-irradiation biopsy ( $\times 545$ ) of residual tumor in right nasopharynx following a tumor dose of 6,610 r. The plasma cells are poorly defined but still recognizable. Several cells show cytoplasmic vacuolation, but the nuclear radiation changes are minimal.

cases it is likely that the lesion is not neoplastic, but a chronic, granulomatous, inflammatory process, with a predominance of plasma cells.

The difficulty of diagnosis microscopically, when the disease involves lymph nodes, is well illustrated by our case, in which the initial diagnosis was reticulum-cell sarcoma. The age of the patient, as well as the distribution of the disease, was compatible with this diagnosis, but the delayed response to roentgen therapy and our subsequent review of the histologic section suggested that the diagnosis was in error.

Treatment of the extramedullary plasma-cell tumors in the past has been chiefly surgical excision for accessible tumors, combined with radiation therapy, or the use of the latter alone. These tumors are radiocurable locally but require intensive therapy, in the order of 5,000 r delivered to the lesion.

The calculated tumor dose delivered to the neck nodes in our case, 4,800 r, appeared to be locally lethal for this plasmocytoma, since the patient was observed for twenty-one months following treatment, without recurrence. A larger dose of 6,610 roentgens delivered to the recurrent tumor in the nasopharynx failed

to eradicate the disease locally. While many factors, such as tumor bed and radiation intensity, may be responsible for this discrepancy, it is also likely that, as with all neoplastic disease, recurrent tumor proves to be less responsive to ionizing radiation than the primary lesion.

#### SUMMARY

A case of primary extramedullary plasmocytoma of the nasopharynx, occurring in an 18-year-old white male, is reported. The disease was of low grade but definite malignancy, with metastasis to the regional lymph nodes and distant multiple osseous involvement.

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#### BIBLIOGRAPHY

1. MATTICK, W. L., AND THIBAUDEAU, A. A.: Extramedullary Plasma-Cell Tumors of the Upper Air Passages. *Am. J. Cancer* **23**: 513-521, March 1935.
2. HAINES, M.: Metastasizing Plasma-Cell Tumour of the Pharyngeal Tonsil. *J. Laryng. & Otol.* **57**: 264-269, May 1942.
3. FRANK, I.: Plasmocytoma of Tonsil. *Ann. Otol., Rhin. & Laryng.* **51**: 22-28, March 1942.
4. HELLWIG, C. A.: Extramedullary Plasma Cell Tumors as Observed in Various Locations. *Arch. Path.* **36**: 95-111, July 1943.
5. FIGI, F. A., BRODERS, A. C., AND HAVENS, F. Z.: Plasma Cell Tumors of the Upper Part of the Respiratory Tract. *Ann. Otol., Rhin. & Laryng.* **54**: 283-297, June 1945.

#### SUMARIO

##### Plasmocitoma Maligno de la Nasofaringe: Un Caso de Mieloma Múltiple Primario

Comunicase un caso de plasmocitoma extramedular primario de la nasofaringe, en un varón blanco de 18 años. La enfermedad era de poca, pero bien definida, malignidad, con metástasis en los ganglios linfáticos regionales e invasión ósea múltiple, remota.

## Prenatal Estimation of Birth Weight By Pelvicephalometry<sup>1</sup>

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**P**ELVIC MEASUREMENTS by means of roentgen rays are indicated and are usually requested in those pregnancies where some abnormality is suspected which might interfere with a normal delivery. Various methods for such measurements have been devised and a review of the literature shows that in each instance either the original author or others have subsequently modified or simplified the technic in an effort to obtain greater accuracy and to eliminate complicated tables of figures or special equipment.

Presumably, every known method of measuring the diameters of the pelvis, whether upon the x-ray film or upon the patient by pelvic calipers, has some limitations and is subject to some error. Undoubtedly the most accurate measurements obtainable in the living subject are the true linear diameters as calculated by any of the various methods of roentgen pelvimetry with corrections for the distortion caused by projection of shadows onto the film. Many of the radiographic methods of measuring the pelvic diameters are subject to only a small percentage of error in the hands of a person accustomed to using a particular technic. It is not unusual for two or more radiologists, each employing a different procedure, to measure the same pelvis and arrive at the same figures for the pelvic and fetal head diameters.

The methods of roentgen-ray measurement advocated by various authors are based upon different principles and fall into four distinct groups: (1) position and grid, (2) stereoscopic films, (3) comparative measurements, and (4) triangulation. It is not the purpose of this paper to discuss the merits or disadvantages of any method. Some are limited to determining the pelvic

diameters and assume that the fetus is of normal or average size. Others measure both pelvic and fetal head diameters to determine whether or not either or both fall within the limits of normal.

After having used one particular method which we consider reliable for a number of years, and feeling that with our experience we had acquired accuracy in measurements, we began to predict the birth weight of the infant. Inasmuch as our prognoses in measured cases had been satisfactory, we felt that it was in order to furnish this additional information. On some occasions our predictions proved to be extremely accurate, but on others they were highly inaccurate, and this we found reflected unfavorably upon our reports of pelvic measurements. The measurements were actual calculations but the weight predictions were estimates only, based upon several variable factors. It soon became evident, however, that the two were being considered in the same light, with the inference that if the predicted weight were incorrect, the measurements must be wrong also. Physicians who had been receiving our reports of measurements of pelvic diameters along with weight predictions became critical of the former, also, when there was a marked discrepancy between the predicted and the actual weight of the newborn infant.

In an effort to determine whether or not birth weight estimations can be made accurately, we began a study of our records. In all of our cases, the Ball method had been used exclusively and several different assistants and residents had participated in the measurements. Ball and Marchbanks (1) have pointed out that it is advantageous to measure the fetal head and the maternal pelvis in relative terms. This

<sup>1</sup> Presented at the Thirty-third Annual Meeting of the Radiological Society of North America, Boston, Mass., Nov. 30-Dec. 5, 1947.

is accomplished by determining the volume of the spheroid fetal head and comparing it with the estimated volume capacity of the pelvic inlet or outlet, whichever is the smaller. Adequate measurements for computing the above values in milliliters (c.c.) may be obtained from two films, an antero-posterior and a lateral view. From these films, it is sometimes possible, also, to obtain the diameter measurements of the fetal skull, as advocated by others (2, 3). These measurements we have found to be of value in those cases where a reading had to be made immediately from wet films, which could be done if the diameters were not foreshortened by the obliquity of the fetal head in the pelvis.

The compiled data reported in this paper were obtained from a study of the records of all obstetrical patients admitted to the hospital from 1940 through 1946 and from the records of those cases which had been referred to the Department of Radiology for pelvic measurements. More than half of the referred patients had been measured and a report had been submitted to the physician prior to admission to the hospital. It was necessary to disregard reports on patients who had been measured but were delivered outside the hospital, as some of the requested information was not available or, if available, was thought to be unreliable. In view of the fact that all definitely abnormal or borderline measurements are routinely checked by another member of the radiological staff, we have not considered it essential to review the measurements to all cases. However, in those cases where subsequent review has shown that the weight of the infant was not reasonably near the weight prediction, all measurements have been checked. This has also been done in those cases where heavy infants have been delivered through apparently small pelvic diameters. All original measurements have been found to be accurate and allowed to stand as reported. As might be expected, a number of the examinations fall under the general heading of "emergency," having been made after the patient had been in labor

for many hours. All measurements which had been made under such circumstances had been subsequently checked before a final report was rendered. These were not checked again, as we usually find that the technical quality of the films and the accuracy of the measurements in such instances are the same as under ordinary circumstances. It is felt that since numerous interpreters have been involved in the calculations and the personal equation has therefore been removed, any errors present would be consistent and would have no effect upon the final analysis of the material.

No attempt has been made to divide the patients into racial groups in order to study the pelvic types in each group. For one reason, the series is too small, and for another, the population of the community where these patients reside is so intermixed that no true racial characteristic would be predominant. The number of colored patients in the series is negligible from a statistical point of view.

This study covers a period during which 8,083 obstetrical patients were admitted to the hospital and delivered. Roentgen-ray pelvimetry was requested and carried out on 338 of these patients. In 30 the findings were abnormal to such an extent that surgical interference was indicated and advised. In all but one of these cases, a successful section was performed and a living infant delivered. Inasmuch as this study deals primarily with cephalometry and fetometry, the information pertaining to the various means and methods of delivery is not considered essential to the paper but is included only as a matter of statistical record. In the 338 measured cases, there were 214 spontaneous deliveries and in all of these it had been reported that a normal delivery could be expected. There were 76 forceps deliveries, of which a number were in conjunction with caudal anesthesia and therefore cannot be classified as indicated by any of the pelvimetry calculations. In some of the remaining cases, forceps were applied in order to terminate labor and not because of any abnormality of the maternal

pelvis or of the fetal head. Cesarean section was performed in 48 of the measured cases and, of these, 18 did not show sufficient disproportion between the fetal head volume and the pelvic volume capacity to indicate surgery, nor had it been advised. For the period under consideration 158 cesarean sections were done, representing approximately 2 per cent of all obstetrical admissions. Of the 158 patients so delivered, 110 did not have roentgen pelvimetry. By studying the records of this group, as well as of the 18 measured cases in which section was done but not because of the roentgen evidence, we find that surgery was decided upon for such various reasons as "placenta praevia," "abruptio placentae," "toxemia," "previous Cesareans," "thirty days past due," etc.

In those cases recommended for section, it can be assumed that the calculations of the fetal head volume and of the pelvic volume were reliable. The head volume determined from the actual birth weight of the infants confirmed the calculated disproportion between the size of the unmolded head and the measured pelvic diameters, although we recognize that the relationship between head volume and weight is not constant.

In one case where the measurements were abnormal, a section was performed and a dead fetus delivered. Stillbirths also occurred in five of the measured cases with spontaneous delivery. No pelvic dystocia or other abnormal finding had been reported in any of these cases and a normal delivery had been predicted. Cesarean section was performed in every case where interference was advised, with the result that no dead babies were delivered because of failure to operate when indicated, nor were there any stillbirths in cases in which delivery was done through pelvis we had reported as inadequate.

Of the 338 patients who were measured, 253 were primiparae, and among these, 26 (10 per cent) showed sufficient abnormality to justify reporting to the obstetrician that section was advisable. Of the 48 sections performed upon the measured patients, 38

TABLE I: SUMMARY OF CASES STUDIED

Total obstetrical admissions.....	8,083
Total pelvic measurements.....	338
Total cesarean sections performed.....	158
Cases with cesarean section measured.....	48
Primiparae measured.....	253
Primiparae having cesarean section.....	38
Primiparae having pelvic measurements and cesarean section.....	26
Stillbirths in measured cases.....	6
Stillbirths, normal delivery.....	5
Stillbirths, cesarean section.....	1

were performed upon primiparae, 26 being indicated by the roentgen findings and 12 for other reasons (Table I). In only 4 cases in the entire series was section advised in a multipara.

In order to determine the percentage of accuracy within which birth weight can be predicted from the calculated fetal head volume, a tabulation was made of the actual birth weight, compared with the predicted weight in each of the measured cases. No weights had been recorded for the six stillbirths which occurred in this

TABLE II: BIRTH WEIGHTS OF MEASURED BABIES COMPARED WITH BIRTH WEIGHT OF 2,000 CONSECUTIVE NEWBORN BABIES UNMEASURED

Birth Weight (lb.)	Measured Babies No.	Per Cent	2,000 Unmeasured Babies No.	Per Cent
2 <sup>1</sup> / <sub>2</sub> -3	0	0	1	0.05
3 <sup>1</sup> / <sub>2</sub> -4	0	0	3	0.15
4-4 <sup>1</sup> / <sub>2</sub>	1	0.29	7	0.35
4 <sup>1</sup> / <sub>2</sub> -5	7	2.05	23	1.15
5-5 <sup>1</sup> / <sub>2</sub>	8	2.34	27	1.33
5 <sup>1</sup> / <sub>2</sub> -6	28	8.19	79	3.95
6-6 <sup>1</sup> / <sub>2</sub>	36	10.53	143	7.15
6 <sup>1</sup> / <sub>2</sub> -7	55	15.09	265	13.25
7-7 <sup>1</sup> / <sub>2</sub>	67	19.59	393	19.65
7 <sup>1</sup> / <sub>2</sub> -8	51	14.91	354	17.70
8-8 <sup>1</sup> / <sub>2</sub>	41	11.99	315	15.75
8 <sup>1</sup> / <sub>2</sub> -9	16	4.68	200	10.00
9-9 <sup>1</sup> / <sub>2</sub>	17	4.97	103	5.15
9 <sup>1</sup> / <sub>2</sub> -10	10	2.92	60	3.00
10-10 <sup>1</sup> / <sub>2</sub>	4	1.19	16	0.80
10 <sup>1</sup> / <sub>2</sub> -11	0	0	9	0.45
11-11 <sup>1</sup> / <sub>2</sub>	0	0	1	0.05
13-13 <sup>1</sup> / <sub>2</sub>	0	0	1	0.05

series. In more than half of the cases, the pelvic measurements had been made and the weight predicted in advance of the anticipated delivery date or admission to the hospital. In such cases it had been necessary to calculate the fetal head volume for the date of expected delivery in order to determine whether or not there would be

TABLE III: HEAD VOLUMES DELIVERED THROUGH Pelves of VARIOUS CAPACITIES, WITH CORRESPONDING BIRTH WEIGHTS

Pelvic Capacity from Smallest Diameter (ML.)	No. of Cases	Measured Cesareans	Largest Head Volume (Calculated) at Term Delivered Through Pelvis and Corresponding Birth Weight				
			Head Vol. (ML.)	Disproportion (ML.)	Birth Wt. (Lb.-Oz.)	Estimated Wt. (Lb.-Oz.)	Error (Lb.-Oz.)
275	1	3	.....Delivered by cesarean section				
325	3	1					
350	1	1					
375	1	1					
400	22	7	690	290	6-13	8-10	1-13
425	4	3	.....Cesarean section				
450	35	10	700	250	6-15	8-11	1-12
475	9	3	625	150	9-13	7-14	1-14
500	3	0	625	125	7-6	7-14	-8
525	86	13	740	215	9-12	9	-12
550	5	0	560	10	8-7	6-10	1-13
575	10	1	590	15	8-2	7-1	1-1
600	65	3	780	180	10	9-5	-11
625	7	0	640	15	7-12	8-2	-6
650	13	1	710	60	9-13	8-13	1-
675	4	0	660	None	7-13	8-4	-7
700	45	1	775	75	7-11	9-3	1-8
750	5	0	690	None	6-14	8-10	1-12
775	1	0	575	None	7-2	6-12	-6
800	10	0	780	None	9-10	9-5	-5
850	1	0	620	None	7-3	7-14	-11
900	5	0	730	None	8-8	8-15	-7
1000	1	0	690	None	8-9	8-10	-1
338		47					

any disproportion between the head volume and the pelvic capacity at that time. The discrepancies between the birth weight figures and the predicted weights have been no greater in those cases where the head volume was calculated prior to term than in those cases which were measured at term.

In calculating the head volume for the time of delivery, the graph published by Ball (4) showing the volume increase in the fetal head in relation to weeks of gestation was used. This curve is considered reliable to within 10 per cent in cases examined within ten weeks of delivery. Scarpellino (5) has devised a similar curve, which we have used as a check in certain cases, but we have not found it to be as accurate for determining the increase in head volume as the Ball graph. From the figures obtained from the graph, showing the estimated head volume at the time of delivery, the mean circumference was derived and upon this the prediction of the body weight was based, tables prepared by Ball (1) being used for both purposes.

A comparison of the actual birth weights in the measured cases with the actual birth weights of 2,000 consecutive newborn infants in the same hospital shows approximately the same percentages of different weights in the two groups, indicating that the infants for whom the predictions had been made were not of unusual size (Table II) and lending support to those radiologists who direct their pelvimetry toward the pelvic diameters only, assuming a fetus of average weight. This finding also suggests that in most instances examination was not requested because the physician anticipated difficulty in the delivery of a large baby. All of the four babies in this series above 10 pounds in weight were delivered spontaneously. In the analysis of the final figures, an attempt was made to determine whether any significant findings were present in the fetal head measurements from which a prediction of the sex could be made. No information could be gained from the data available to justify any conclusion on this point.

Estimated birth weight is added informa-

TABLE IV: BIRTH WEIGHT AT DELIVERY WITH CORRESPONDING HEAD VOLUME AND PELVIC CAPACITY

Smallest Pelvic Diameter (Cm.)	Capacity from Diameter (Ml.)	Heaviest Baby Delivered Through Pelvis and Corresponding Head Volume					
		No. of Patients	Head Vol. (Ml.)	Disproportion (Ml.)	Birth Wt. (Lb.-Oz.)	Estimated Wt. (Lb.-Oz.)	Error (Lb.-Oz.)
8.25	275	1					
8.5	325	3					
8.75	350	1					
9.00	375	1					
9.25	400	22	600	200	9-7	7-10	1-15
9.3	425	4					
9.5	450	35	660	210	9-10	8-5	1-5
9.75	475	9	575	100	9-15	6-12	3-3
9.9	500	3	515	15	7-15	6-3	1-12
10.00	525	86	610	85	10-4	7-12	2-8
10.15	550	5	440	..	8-8	5-8	3-
10.25	575	10	560	..	8-13	6-11	2-2
10.50	600	65	600	..	11-3	7-10	3-9
10.6	625	7	630	5	9-5	8-2	1-3
10.75	650	13	710	60	9-13	8-13	1-
10.9	675	4	640	..	9-9	8-2	1-7
11.00	700	45	705	5	10-6	8-12	1-10
11.25	750	5	610	..	9-2	7-12	1-6
11.35	775	1	575	..	7-2	6-12	-6
11.50	800	10	780	..	9-10	9-5	-5
11.75	850	1	620	..	7-3	7-14	-11
12.00	900	5	730	..	8-8	8-15	-7
12.5	1000	1	690	..	8-9	8-10	-1

tion for the obstetrician, especially in the multiparous patient, as the adequacy of the pelvis has already been established to a certain extent by the birth weights in previous deliveries.

The cases in this series have been tabulated to show the largest head volume delivered through the various pelvic diameters with corresponding birth weight (Table III) and the heaviest baby delivered through a pelvis of similar diameter with the corresponding head volume (Table IV). The discrepancy between the head volume from which the weight is predicted and actual birth weight does not have the same significance as the difference between head volume and pelvic capacity. For example, a minimal pelvic diameter may allow passage of a greater birth weight than predicted from the head volume but that same diameter will not allow passage of a head from which such a birth weight would be predicted. All of the cases having the same measured head circumference received the same figures to denote head volume and predicted body weight. Actual birth weight, however, when compared to the predicted weight showed that there was a wide variation among those having the

same head volume. These variations cannot be explained by sex difference nor can they be explained by the probable maximum error of 10 per cent in calculating spheroid volume from mean perimeter measurements.

Seeking further information to explain the differences in birth weight in infants with the same head volume, another group was studied. Thoms (2) has shown the relation to fetal length of the occipitofrontal diameter. With this relationship in mind and the idea that a probable explanation was the general body build and development of the fetus, a series of consecutive newborn infants was studied. Measurements were made of the head circumference and body length within twenty-four hours after birth. It was found that two or more babies with precisely the same head circumference and of the same sex would be of different length and have a different birth weight (Table V). Birth weight predictions from head volume must of necessity depend upon a number of variable factors. The prenatal growth curves which have been computed by different authors vary considerably, and the estimated time of delivery may be

TABLE V: BIRTH WEIGHT AND LENGTH IN RELATION TO HEAD VOLUME AND ESTIMATED WEIGHT

Mean Circumference (Cm.)	Head Volume (Ml.)	Estimated Weight (Lb.-Oz.)	Average Weight (Lb.-Oz.)	Lightest Weight (Lb.-Oz.)	Heaviest Weight (Lb.-Oz.)	Shortest Body Length (Cm.)	Longest Body Length (Cm.)
31.50	530	6-7	6-8	6-6	6-10	52.07	52.07
32.00	560	6-10	5-8	5-3	5-13	48.25	49.50
32.50	580	6-13	7-2	6-9	7-12	49.50	53.34
32.75	592	7-4	6-12	5-7	8-0	48.89	53.34
33.00	605	7-10	7-1	6-9	7-8	49.50	52.07
33.50	640	8-2	7-3	6-9	7-13	48.25	53.34
34.00	670	8-6	7-12	6-12	8-12	48.25	55.88
34.50	690	8-10	7-12	7-5	8-3	48.25	53.34
35.00	725	8-14	8-15	7-15	9-14	52.07	57.15
35.50	760	9-2	9-3	8-12	9-9	53.34	53.34
36.00	800	9-7	9-11	9-7	10-0	52.07	54.61

wholly erroneous or, even if it be correct, labor may begin earlier than expected. These factors must be taken into consideration as accounting in part for the relative inaccuracy of such prediction when the pelvimetry is done well in advance of the birth date. Using the Ball curve for growth *in utero* and combining those estimations with the predictions made at term and during labor, we have been able to predict birth weight to within 4 oz. in 22 per cent of the cases, to within 8 oz. or less in 36 per cent, and have failed to predict to within 8 oz. in 62 per cent, while in 2 per cent no predictions were made (Table VI). These incorrect predictions obviously are not the result of faulty roentgen technic or inaccurate measurements, since the pelvic diameter measurements upon these patients were verified as correct by the outcome of labor.

From the point of view of the obstetrician, the prediction of birth weight is in most cases of slight significance. He is not perturbed about those women with adequate pelvis according to his clinical measurements nor is he interested in radiologic consultation in those cases where there is obvious evidence of an abnormal pelvis. Roentgen pelvimetry is then of the greatest importance in the borderline cases for discovering anatomical variations in the maternal pelvis, abnormal position of the fetus, or any condition which can be more accurately evaluated by this method than by external pelvimetry. The type of the pelvis, the shape of the sacrum, and the obliquity of the pubic angle should always

be given consideration, in conjunction with the measurements, in order to complete any roentgen pelvimetry report.

By employing the Ball nomogram in this series of cases, it has been possible to calculate the fetal head volume from the head circumference. The accuracy of these corrected measurements has been verified by

TABLE VI. PERCENTAGE OF ACCURACY IN BIRTH WEIGHT PREDICTIONS

Total pelvic measurements.....	338
Total delivered babies alive.....	342
Predicted weight varying 0-4 oz. from birth weight.....	74 (21.6%)
Predicted weight varying 4-8 oz. from birth weight.....	49 (14.3%)
Total 8 oz. or less.....	123 (35.9%)
Predicted weight varying 8-12 oz. from birth weight.....	45 (13.2%)
Predicted weight varying 12-16 oz. from birth weight.....	44 (12.8%)
Predicted weight varying 1-1½ lb. from birth weight.....	60 (17.8%)
Predicted weight varying 1½-2 lb. from birth weight.....	35 (10.1%)
Predicted weight varying 2 lb. or more from birth weight.....	29 (8.5%)
Total varying from 8 oz. plus to 2 lb. or more.....	213 (62.4%)
No prediction of birth weight.....	6 (1.7%)

measuring the head of the newborn infant. Other observers (6) have found this method of measuring the fetal head to be accurate to within 1 cm. Although the method may not be an exact procedure, it is sufficiently accurate to indicate that both circumferences were measured to within 10 per cent when calculated into milliliters head volume. The variations disclosed between actual birth weight and the weight predicted from the head volume

have shown more than the probable 10 per cent error. This difference in weight is not the result of faulty measurements or of the methods of calculation, but can be explained only by the difference in muscular, skeletal, and fatty development at the time of birth. Measurement of the fetal head by roentgen cephalometry is as accurate as the measurements of the pelvic diameters. Fetal head diameters and circumferences can be obtained, and from these head volume can be calculated accurately. These measurements and calculations show the size of the fetal head in comparison to the pelvic diameters as an aid in solving an obstetrical problem. The fetal head size is at the present time the best known index for estimating birth weight, but other unmeasurable factors may be present to such an extent that such estimations will be entirely erroneous and misleading and thereby discredit the value of the original examination of which the predicted birth weight is merely a by-product.

#### CONCLUSIONS

1. Cephalometry is the only basis upon which birth weight can be predicted with any degree of accuracy.
2. The birth weight of an infant can be predicted from the fetal head volume to within 8 oz. in 36 per cent of cases and to within 16 oz. in 62 per cent.
3. The inability to predict the actual weight within 8 oz. in 2 out of 3 cases renders the process of doubtful practical value.
4. The fetal head size or volume is more important than the body weight

when considering delivery through pelvic diameters.

5. Skeletal, fatty, and muscular development are not measurable *in utero*, and variations in these influence birth weight more than the head circumference.

6. Incorrect prediction of birth weight from head volume is not due solely to faulty technic or to inaccurate measurements.

7. Previous birth weights in multiparous women are not absolute proof of the adequacy of pelvic diameters.

8. Abnormal findings in primiparae in this study are not sufficient to warrant routine pelvimetry on all primiparae.

9. Inaccurate birth weight predictions should not in any way discredit the value or the accuracy of roentgen cephalometry or pelvimetry.

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#### REFERENCES

1. BALL, ROBERT P., AND MARCHBANKS, S. S.: Roentgen Pelvimetry and Fetal Cephalometry: A New Technic. *Radiology* **24**: 77-84, January 1935.
2. THOMS, HERBERT: Cephalometry *in utero*: Method for Estimating Occipitofrontal Diameter, and Statistical Study of Cephalic Measurements in 149 Unmolded Heads. *J. A. M. A.* **95**: 21-24, July 5, 1930.
3. DIPPEL, A. LOUIS, AND DELFS, E.: Accuracy of Roentgen Estimates of Pelvic and Fetal Diameters. *Surg., Gynec. & Obst.* **72**: 915-921, May 1941.
4. BALL, ROBERT P.: Roentgen Pelvimetry and Fetal Cephalometry. *Surg., Gynec. & Obst.* **62**: 798-810, May 1936.
5. SCARPELLINO, LOUIS A.: Cephalopelvimetry. *Radiology* **48**: 45-50, January 1947.
6. WEINBERG, ARTHUR, AND SCADRON, SAMUEL J.: The Value and Limitations of Pelvioradiography in the Management of Dystocia, with Special Reference to Midpelvic Capacity. *Am. J. Obst. & Gynec.* **52**: 255-263, August 1946.

## SUMARIO

## Apreciación Prenatal del Peso del Feto por medio de la Pelvicefalometría

En una serie de 8,083 ingresos obstétricos consecutivos en un hospital, se ejecutó la roentgenopelvimetría en 338. Calculóse el volumen de la cabeza del feto conforme al método de Ball, y por el mismo se computó el peso del cuerpo de acuerdo con las tablas facilitadas por Ball (*Radiology* 24: 77, 1935). A fin de determinar el porcentaje de exactitud con que puede predecirse así el peso al nacer, se comparó el peso real obtenido con el calculado en todos los casos medidos. Las predicciones resultaron correctas con una aproximación de 120 gm. en 23 por ciento de los casos y con una aproximación de 240 gm. o menos en

36 por ciento. Este resultado pone en duda el valor de tales predicciones. Los errores no se deben a defectos de la técnica o a inexactitud en las mediciones, sino a diferencias en el desarrollo esquelético, adiposo y muscular que no pueden medirse en el útero.

El tamaño o volumen de la cabeza del feto es más importante que el peso del cuerpo al considerar el paso por una pelvis de diámetros dados, y las predicciones inexactas del peso no deben en modo alguno desacreditar el valor o exactitud de la roentgenocefalometría o pelvimetría.



## A Symposium on Socialized Medicine

Presented at the Thirty-third Annual Meeting  
of the Radiological Society of North America

### Introduction<sup>1</sup>

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FOR A GREAT MANY years there has been an increasing tendency all over the world for governments to intervene in the practice of medicine. Such intervention almost regularly results in government control of the practice of medicine. This whole movement goes back a very long way, originating in 1884 in Germany, when the Iron Chancellor, Bismarck, organized the first German social insurance system to prove to the Labor Party how much love the government had for them and how solicitous it was of their welfare.

We are almost the last great country of the world where medicine still remains free enterprise and where it is not dominated by government-controlled agencies. How much longer we shall be, depends in no small measure upon us, ourselves. If we are going to cope with any problem of any sort, we must first of all understand the problem. It is my intention now briefly to summarize the problem for you, and to tell you what it is that is said by the proponents; I will leave it to our able essayists to refute them. Very frequently I have done this, and later people have said that I was a proponent of socialized medicine, so I direct your attention carefully to the fact that I shall be saying not what I think but what the people who want socialized medicine think, or say they think.

Socialized medicine is an awfully loose term; it really means compulsory sickness insurance to be enacted by law. The pro-

ponents of this plan say that the state of the health of the American people is indeed pitiful; that unnecessary disease is rampant everywhere; that the number of cases of medical neglect is appalling; that only the rich man can get any medical care; that the poor must ask for charity, and that those that lie between simply can't get charity or medical care.

They say that the distribution of doctors is extremely inequitable; that there are large areas of this country in which medical care is not available; that there are many counties in which there is no hospital.

They say that these conditions could be healed by the enactment of a compulsory sickness insurance law. They point with great horror to the number of people who were rejected in our draft during the last war, the five and a half million 4-F's, and they say that this astonishing revelation indicates, as nothing else has ever been able to indicate, the appalling state of health of the American people.

If we could just enact compulsory sickness insurance, we could cure all these ills. Then everyone would get medical care for a trifling amount of money; the amount of money is relatively unimportant and, as a matter of fact, it is simpler not to talk about it, because it's much easier to pass a bill without money appropriations than it is to pass a bill with money appropriations, and it is perfectly obvious that if Congress enacted a bill to provide for compulsory sickness insurance, it would be silly to

<sup>1</sup> It was a source of regret to all that the Rev. Alphonse Schwitalla, S.J., Dean of the School of Medicine, St. Louis University, who was to have opened this Symposium with a discussion of "Socialized Medicine and Its Effects on the Hospital and Medical School," was critically ill and unable to present his paper. In Father Schwitalla's absence, Dr. Lowell Goin, the *Chairman*, who has been an active crusader for the proper relationship of radiologists to medicine and hospitals, presented this brief Introduction.—Ed.

refuse to enact the necessary appropriation.

That is the mechanism by which we approach this plan.

Now, is our health bad? If it is, we ought to consider what to do about it. It doesn't necessarily follow that, if it were bad, the measures proposed would be a satisfactory solution. But, if it isn't bad, a good deal of force of the argument has been lost.

The United States Public Health Service lately issued a report on the incidence of tuberculosis in the world. The United States has not had the benefits of compulsory sickness insurance; we are a benighted country where free enterprise still exists, but the incidence of tuberculosis in the United States is less than 25 per 100,000 of population. Great Britain has had compulsory insurance since 1911 and, by a strange coincidence, their incidence of tuberculosis is stated as between 50 and 75 per 100,000, while in Germany, which has enjoyed these benefits since 1884, the incidence is given as between 75 and 100 per 100,000.

Diphtheria is a good indicator, because it is a specific disease for which we have a specific treatment and, as a matter of fact, a disease from which no one need to die. There are no secrets involved. German and English doctors know just about as much about it as do you, yet in the last year in which there were any comparable figures, the death rate from diphtheria in the United States was somewhat less than 4 per 100,000 of population, while in Germany it was 11.6 per 100,000 and in Great Britain it was 11.7 per 100,000 of population. In this instance, then, the panacea of compulsory health insurance seems to have failed slightly in its stated objective!

Where are these cases of medical neglect that the proponents speak of so glibly? I think it is very strange that none of us ever sees them, though we are doctors that practise medicine and who see the sick. On one occasion, in a radio debate, I denied the existence of such neglect and invited members of the radio audience—which was

estimated at over 100,000—to inform me if they knew of a case of medical neglect and assured them that I would undertake to get them some medical care. Next day I had one call. The man who called me said he was such a case and he could prove it by the lawyer. He had broken his hip; he had been in the Cedars of Lebanon Hospital, one of our biggest and finest hospitals, for thirteen weeks; he had been seen by eleven specialists and had had his hip x-rayed forty-one times and it still wouldn't heal!

The Medical Society of the State of New Jersey conducted a public relations radio campaign for a period of something more than one year, in which they urged any person unable to get medical care to communicate promptly with the executive offices of the Society and undertook to supply medical care to them. In the year they had 84 replies.

Remember, New Jersey is a densely populated industrial state. They had 84 replies, and of the 84 who replied, something like 70 were not people who were unable to get medical care but people who, because of language difficulties and lack of orientation in our particular civilization, simply didn't know how to go about getting it. It wasn't that they couldn't get it; they just didn't know what to do.

In Alameda County, California, the County Association has advertised in all the newspapers for a period of one year making the same offer. Alameda is a large county and contains the large city of Oakland; I suppose that its population is perhaps one and a half million. Yet in the year's campaign there were only six persons who replied. So, I ask again: Where are these cases of medical neglect?

"There are large areas of the country without medical care available." Of course there are. The state of Nevada contains something like 110,000 or 115,000 people and I suppose you could put four states of Massachusetts in it. Well, how shall we solve that problem? Why will compulsory sickness insurance suddenly produce doctors for the state of Nevada?

How many doctors do you need to take care of the people in the state of Nevada? These are questions which are by no means solved by compulsory sickness insurance.

"It is difficult to get doctors to locate in hamlets and villages." Of course it is, and the reason is a very complicated social one. Doctors are intelligent, literate people, well educated; they are accustomed to the society of their peers; they like to have access to hospitals and to libraries; they like to go to medical meetings; they just don't like to go out and live in little villages. Will they like it any better with compulsory sickness insurance? Of course they won't.

The only possible way of getting the doctor to live in the hamlet is the system which has been adopted in England, where the government says it will encourage doctors to locate in small villages and thus give better medical care by a simple device. First, it will pay a somewhat better capitation fee and second, it will forbid them to locate elsewhere.

Mr. Truman said in his message to the Congress that one thing we didn't want was socialization, and that there was no intention of the government to compel anybody to do anything—that is, except to compel every person on a payroll to pay part of his salary in this tax and to compel every employer of persons to pay part of his wages out to the bureau to be set up to receive them. I think it a very strange coincidence that the last Murray-Wagner-Dingell bill contained a section that specifically waived the provisions of the Public Health Act of 1910 that authorized the Surgeon General to commission any number of medical officers he saw fit and to order these men to duty wherever he saw fit. If the Public Health Service intends to practise medicine in the villages of the country, that solves the problem, but Mr. Truman denied that they intended to practise socialized medicine.

As to the draft figures, they have been debunked so many times and so thoroughly that it is amazing that people keep on quoting them. I think it a tribute to the

dishonesty of the proponents of compulsory sickness insurance that they keep on using these thoroughly discredited figures. I debunked them before the Murray Committee two years ago; before the last Senate Committee hearing last June Dr. Friedman did it much better, and it seems incredible that anyone would keep on using them. Yet every address made in behalf of compulsory health insurance quotes these figures as though they had some significance.

The plain fact is that if you look at the experience in the world today, you will find that there is no place where the public health is as good as it is in this country; that there is nowhere that medical science has advanced as much as it has in this country; that the European countries have lost their pre-eminence as educational centers; that the day when the American doctor strove vigorously to get to Germany or Vienna to advance his education has gone and, as a matter of fact, everyone out of this country wants to come here to advance his education. The level of public health is lower in other countries than it is here. In most of them it is much lower.

All experience would persuade any reasonable person looking into the matter that compulsory sickness insurance is an extraordinarily poor way to give medical care. If it is a very poor way to give medical care, and if its general effect in the world is to lower the standards of health rather than to improve them, and to decrease the standards of medical practice rather than to elevate them, why is it that comparatively intelligent and sincere people continue to urge the enactment of such legislation?

To this I am going to give you a very flat answer, which I think will be thoroughly implemented by the next speaker. It is my earnest belief that the proponents of compulsory sickness insurance have but little interest in the health of the American people and that they have a profound interest in the erection of a national socialist state. I think that all too few of us are aware that revolution has now become a department of knowledge, that it has its

literature, its textbooks, its intellectuals, its professors.

These persons have set down the technic for effecting revolution, and among the things to be accomplished are the seizing of physical control of the gold, the debasing of the national currency, the creation of class consciousness and the incitement of racial hatred, and what they very quaintly term the domestication of individualism.

Now ask yourself where the gold is, and what is the value of the dollar now in your pocket. Think back to when you were small children and ask yourselves whether there was any class consciousness. Then ask yourselves whether it has been created. Is racial hatred being stirred up in this country or is it not?

The domestication of individualism is a nice phrase, which is defined as anything that can be done which will render the citizen more subservient to the State. Some years ago I sat in the House of Delegates of the American Medical Association at Atlantic City and heard Senator J. Hamilton Lewis make the statement—which so shocked me that I have never for-

gotten it: "We are compelled to tell you that we do not recognize such a thing as the physician and patient; we do recognize an entity called the citizen, a creature of the State." (Note the natural superiority of the American statesman.)

Hitler wrote a large, dull book to set forth that theory so neatly stated by Senator Lewis in two sentences. All American theory is that the State is the creature of the citizen—that we can overturn it or put rascals out of office—but under this theory we become the State's creatures and its subjects. This is the goal and objective of the compulsory sickness insurance proponents. As I say, I think that our next speaker will well implement it for you, because she is an extraordinary woman—extraordinary in intelligence, with an extraordinarily wide experience in this whole matter, formerly technical adviser to the minority group of the United States Senate which since, by God's grace, has become the majority group—Dr. Marjorie Shearon.

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## Government Interference in Medicine

MARJORIE SHEARON Ph.D.

I am very glad to have this opportunity to speak to a group of this size and representation throughout the country on the subject of "socialized medicine." I wish I could get into every little hamlet and speak to groups of doctors, business men, and citizens who are not professional or business men. I have just returned from a 15,000-mile trip on which I have spoken before forty different audiences in fourteen states. I wish I had time to continue that sort of thing. I feel it is very necessary. The medical profession, business men, and other groups have no idea how close this thing is upon them. The time is later than you think.

Remember this: We are approaching a presidential election year and for the first

time in the history of the United States a President has said, "I wish to have medicine socialized." He doesn't put it in those words; he has been told that what is in view is not socialized medicine, but it is. I prefer to use the word "nationalized" because then there can be no mistake about what is meant, namely, control by the Federal Government.

The President of the United States has made it a part of his legislative program, he has asked federal agencies to push for the program and they are doing it as you very well know. You have seen what happened to Dr. Parran in 1944 when he was told to get on the band-wagon of the Wagner-Murray-Dingell bill and he got there instead of standing up and saying that he

didn't approve the bill and compulsory sickness insurance. He didn't have the courage to do that.

The members of the Social Security Administration, the Public Health Service, the Department of Agriculture, the Department of Labor—all are on the bandwagon putting forth propaganda the like of which we haven't seen before. Probably most of you know that a committee in the House of Representatives has been investigating this propaganda. In a unanimous report, signed by Republicans and Democrats, the House Committee on Expenditures has said that such propaganda exists and has asked for its investigation by the Department of Justice. During the summer the Department of Justice looked into the situation and prepared a report on its own account asking the Federal Bureau of Investigation to investigate these federal officials who have been breaking the law against lobbying.

The Federal Bureau of Investigation has nearly completed its report. If they ask for the prosecution of these officials, the case will be tried in the criminal court of the District of Columbia and officials who are found guilty will be liable to a year in prison and/or a \$500 fine.

This is serious business. For the first time these lobbying activities of federal officials have been recognized officially, although there were plenty of us inside the government who knew it years ago, and there were plenty on the outside who suspected the worst. Now at last there has been official recognition by a committee in Congress and an official investigation by the Department of Justice and by the F.B.I. I can only hope that there won't be any politics in the end that will keep that investigation under cover or will attempt a job at whitewash.

The hearings on bills S-545 and S-1320 will be resumed in January. There are not supposed to be very many more witnesses. As you know, Mr. Falk, who has written or has supervised the writing of all these so-called health bills since 1939, will be recalled to be cross-examined in great detail

by Senator Donnell. I have been asked by Senator Donnell to appear myself as a witness. Instead of being a consultant to the Senator, I shall step down and be a witness. I intend to do so and to bring out to the best of my ability the whole story of the movement to nationalize medicine.

I want again to call your attention to the fact that this is now a major political issue. For the first time in the history of this country a President of the United States has asked Congress to enact a bill for national compulsory "health" insurance. Although by and large the Democrats have been the ones who have sponsored this legislation, you cannot count on this being purely a Democratic move. Consider what would happen if Senator Taft and Governor Dewey were to liquidate each other and disappear from the picture as possible presidential candidates for the Republican nomination, and Governor Warren of California should show up as the person who might get the Republican nomination and perhaps become the Republican President.

Governor Warren has been sold the bill of goods on compulsory sickness insurance. He has tried to sell a miniature Wagner-Murray-Dingell bill in California. You would then have a compulsory bill as a Republican measure instead of its being, as at the present time, predominantly a Democratic measure. You cannot therefore count on a compulsory bill being confined to one party.

I want to spend a little time on S-1320, providing for medical care insurance. This may sound like rather dull stuff but, believe me, it's the thing that the medical profession needs to go into. Study that bill. Go back, furthermore, to S-1050 in 1945 and S-1611 in 1943 if you wish to see the total blueprint for the nationalization of medicine, and remember that that is a small part of the over-all nationalization scheme.

Countries do not socialize medicine in a vacuum. They work for the socialization of the entire economy, as they are doing in

Great Britain. That was Bismarck's plan; it is still the plan today in this country and in other countries.

The need for federal intervention in the provision of health and medical services for the entire population is being urged upon the Congress of the United States on the ground that present inadequacies and lack of availability of health services are such that the preservation and improvement of the health, vigor, and security of the American people are being impaired. Its proponents claim further that a National Health Program is required not only for the general welfare but as a measure of common defense and national security. Note that well. There is a gradual transition from saying that federal intervention in the field of medical care is for the general welfare; they are slipping over to the statement that it is for national security. They will have a better chance of achieving it "for national security," for the defense of the country, than they will if they say simply "for the general welfare."

The objectives of such a National Health Program are so worded as to be all things to all men. Do read the preamble of the Wagner-Murray-Dingell bill if you want to see the promised millenium! The program which the Congress is to establish is not only "to aid and foster health and medical progress throughout the Nation" but also "to prevent sickness, disability, and premature death." It is "to promote personal relationships between physicians and patients...to stimulate scientific advancement" and "research...to enable patients to have more effective free choice in selecting their physicians, and...to be administered locally...in a manner designed to preserve the customary freedom and responsibility of professional persons in the exercise of professional judgment as to the care of a patient."

I have just read part of the preamble to the Wagner-Murray-Dingell bill; these are the noble objectives that are stated. These and other laudable purposes are the *avowed* objectives of this latest compulsory sickness insurance bill. The Congress, organ-

ized labor, and the public at large are led to conclude that the substantive provisions of the bill would lead to fulfillment of its declared purpose. Let us examine the objectives and then ask ourselves whether the proposals set forth in the Wagner-Murray-Dingell bill are likely to bring about the results they are allegedly designed to achieve.

It is important to realize the wide divergence between the stated objectives and the actual provisions of the legislation, because a lot of people are being sold down the river by the objectives without having the slightest notion about the contents of the bill. They have not seen the complete divorce between what the bill is really going to do and what it *says* it is going to do.

I want to illustrate the way in which the law would operate by just two or three provisions in the bill itself. Let us consider, for instance, the avowed purpose of *promoting personal relationships between physicians and patients*. How would this be done?

Well, first we note that every physician, after enactment of the law, would be held responsible not solely to his patient, as he is at present, but jointly *to the State and to the patient*, the State presumably having precedence over the patient in this regard. That is in the Wagner-Murray-Dingell bill, Section 216 (c).

Secondly, under the Wagner-Murray-Dingell bill, a patient normally may consult a specialist only with the permission of the government insurance doctor. In other words, at the present time a patient may go to a specialist if he feels like it. It is his own responsibility. He may carry out his own desires or the wish of his family in this respect. Under the Wagner-Murray-Dingell bill he could not normally do so unless he had the okay of the insurance doctor. Of course, if he could not get the permission of the insurance doctor, he could appeal to the government referee or, as he is called, "the administrative medical officer," that is, the government official to whom patients and doctors would be referred if they were dissatisfied with any-

thing. Such officers would be charged with responsibility for policing the "health" insurance system.

If the Federal Government, through regulations to be written by a National Health Insurance Board, decides to permit a patient to call in an unauthorized specialist, the government *may* do so, but nothing in the law would guarantee to a patient the right which he or a member of his family now exercises of summoning a specialist. Think what that would mean, for instance, if a patient felt that he had a disease that warranted consultation with a specialist. He can have such consultation now, and if he happens to have a general practitioner who isn't too alert, who hasn't sensed the needs of the case, there is some recourse. Under the Wagner-Murray-Dingell bill there would be no such recourse, because the government doctor could say: "You may not go to a specialist."

Of course, the patient could appeal, but any of you who have ever appealed anything to the government know that the patient would probably be dead before he could get the specialist. I doubt if this interposition of the government as represented by a state agency or by a National Health Insurance Board or by a federal referee between a patient and his physician will promote their personal relationships as is claimed in the preamble of the bill.

And I am quite sure that the patients of this country haven't the slightest notion in the world that this is what is actually written into the bill and would become law if the Wagner-Murray-Dingell bill were enacted. There is not a single person that I have spoken to who, when I have mentioned this fact, fails to say, "Oh my goodness! Is that in the bill? I don't want anything like that."

I believe, as a matter of fact, that the provisions in this bill would not only *not* improve the relationships between patients and their physicians but would create great friction and that the life and peace of mind of the patient would be jeopardized in many instances. I cite this as one illustration of the fraud that is being perpe-

trated through this bill and of the false propaganda in connection with it. The bill has rightly been called, at the Senate hearings, "a fraud on the American people."

Another laudable purpose of the national health legislation referred to is the patient's freedom of choice of physician. *Maximum freedom* is obtained when a patient is at liberty to go to any physician regardless of geographical and other limitations and when services are paid for on the basis of a fee which relates to the quantity and quality of the care given by the practitioner. *Minimum freedom* would be obtained if a patient were to be compelled to accept services from a single salaried physician, as might be the case in a rural community having only one practitioner.

I have just come from Hawaii, where they have a single salaried doctor taking care of the entire population on a plantation. There is no freedom of choice. I am not condemning that form of medical practice in Hawaii because I think it serves its purpose very well there. I cite it simply as an illustration of a salaried service without freedom of choice.

An intermediate condition between full freedom and none would be partial freedom under a capitation system. It is important to note that the representatives of the two leading non-governmental lobbies, namely Michael M. Davis of the Committee for the Nation's Health, and Ernst P. Boas of the Physicians' Forum, have both conceded that *the fee-for-service basis of payments will have to be abandoned under national compulsory sickness insurance.*

Davis stated under cross-examination that the capitation method of payment would be the one which would probably be adopted. The long-range effect, if the capitation system is followed, is twofold: (1) It levels the salaries of physicians and surgeons downward and (2) it greatly restricts freedom of choice by patients and practitioners alike.

Dr. Boas has made the same admission, stating that you couldn't possibly have compulsory sickness insurance for the entire population and still retain the fee-

for-service method of payment. The Wagner-Murray-Dingell bill contains the provision that payment would be by fee-for-service, capitation, or salary, or by a combination of those three methods. That was put in simply as a come-hither to the medical profession and to patients, to make them think that the present method of payment would remain. But the sponsors of the bill know perfectly well, behind the scenes, that it couldn't remain, administratively. It would break the system financially. It couldn't be done.

Even in a little country like New Zealand, which has gone broke in six years under a comprehensive system of compulsory national social insurance, and in which the medical care program is in a chaotic condition, they have found they cannot use a fee-for-services basis. It is impossible, but the medical profession at large isn't told that and we still have the propaganda coming out of Washington which says, "Why, everything will be just the same as it is now. The only difference will be that the government will pay the bill instead of the patient having to do so at the time the service is rendered."

The protagonists of this legislation know, when they put out that sort of information, that it isn't true and that they don't intend it to be that way and that they couldn't work it if they tried to do it. It would fail.

The Wagner-Murray-Dingell bill furthermore contemplates the pro rata assignment of recalcitrant patients to physicians not of their own choosing if, in a capitation area, some persons refuse to pick a physician. In other words, we must realize that if this legislation is enacted we are likely to have a capitation or panel system such as they have had in Great Britain for 40 per cent of the population. Great Britain had that system for thirty-five years. Now they are threatened with the nationalization of medicine.

So we have to ask, "What are some of the effects of a capitation system?"

One is that patients who fail to choose a physician have to be prorated, since the

doctor's income is dependent upon the number of patients on his list. If, say, you have a hundred doctors in a community and you have a 100,000 population and the law says that each doctor may have 1,000 patients, then each doctor *must* have about 1,000 patients in order to get his income. What are you going to do if a certain number of patients say they are not going to get on any doctor's list?

The Wagner-Murray-Dingell bill has stated that after a certain period, say three months, people would be notified that they would be assigned—and *they would be assigned*. Is that freedom of choice, and would it improve the relationship between the patient and the physician to be assigned? Yet it is an inevitable corollary of a capitation system where patients are worth so much per head per year to the physician on whose list they appear. They are simply federal pawns and they are moved around like pawns, but the people are not being told this, and I don't think most of the doctors understand that this is the plan.

One of the points which has been greatly stressed regarding S-1320 is that it is "to be administered locally in accordance with American ideals of democracy and individual freedom." I quote from the bill. That has been one of the keynotes of the propaganda this year, namely: "There won't be anything centralized in Washington; it will be administered locally."

If you will go through the bill you will find that there is no such intention. Whatever local committees are set up, either of the profession or of the public at large, may operate only under the rules and regulations from Washington and they must agree, if they come in under the state plan, which is approved in Washington, to accept the rules and regulations from Washington. We would therefore not have local administration but only talk about it, as in the bill.

Now I want to refer to the three methods by which power would be centralized solely in Washington and solely in the hands of the Federal Security Administrator. The

bill states that there shall be set up a National Health Insurance Board of five members, three of whom are to be appointed by the President and the other two of whom shall be the Surgeon General of the Public Health Service and the Commissioner of Social Security—at the present time Dr. Parran from Public Health and Mr. Altmeyer. (You can visualize these things more easily if you get the names of people who are now incumbents.) The other three persons would be appointed by the President. Only one needs to be a practising physician, and you can jolly well bet that the other two wouldn't be.

So you'd have a National Health Insurance Board of which only one member would be a practising physician. The Surgeon General of the Public Health Service isn't required by law to be a physician and the Commissioner of Social Security, I presume, would never be a physician.

That is the all-powerful Board. It carries on the business of the whole social insurance scheme which is designed to cover the entire population, and no one would be left out; even *you* couldn't stay out. All workers would be taxed and all persons eventually would be brought in.

That National Health Insurance Board is then set up by the President; working with it, but not doing very much work, is the National Advisory Medical Policy Council of seventeen members. Without going into details on that, it is *not* a Medical Policy Council. That is the name that is given to it because it sounds well. It would not have a majority of medical people on it; in fact, it might not have more than one or two physicians. It doesn't have to meet more than twice a year and it doesn't meet unless it is called by the National Social Insurance Board.

This Board is to administer the program "under the direction and supervision of the Federal Security Administrator." That is what the bill says. *The final authority, therefore, for this entire program is vested in*

*the Federal Security Administrator, who is a political appointee without any knowledge in the field of health and medicine.* The present incumbent is Oscar R. Ewing, who was promised the job with the expectation that he would get a Cabinet post out of it when and if the bill is enacted which would elevate the Federal Security Agency to Cabinet status (S-140).

If you will read the W-M-D bill you will find that this top administrative Social Insurance Board is mentioned fifty-six times in the sixty-three pages that deal with compulsory sickness insurance. It is the Board authorized to do this and that, it decides, it does so-and-so; it runs the health and medical professions of this country, including the doctors and dentists, the nurses, the hospitals, the technicians, the radiologists, and all the other people who contribute to the provision of medical care, hospital services, and so on.

If you really saw the picture, then, of this legislation and of the authority given to lay people to run the medical program of this country, you would be much more up in arms about it than you are at the present time.

Finally, I want to say that the present organization in the Federal Security Agency is heading up in preparation for the enactment of this bill. Mr. Altmeyer, who is the Social Insurance Commissioner, has now been made the Deputy *pro tem* to the man at the higher level, namely, the Federal Security Administrator. He has now been raised from being just the head of Social Security to the head of Social Security and Health and Education. The administrative set-up is being prepared and the ground work is being laid for enactment of this bill (S-140) during the coming year in accordance with the wishes of the President. I warn you to be on the alert with regard to these and related bills designed to legalize government interference in medicine.

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(Symposium continued on following page)

## The Implications of Social Medicine For Radiologists

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IN THE VERY BEGINNING, I should like to consider the title assigned to me in this symposium. In doing so, I shall indulge in some semantical acrobatics. The semanticist is concerned with the simplification of words of high abstraction in such a manner as to establish a common understanding among all people as to their true meaning and to eliminate emotional reactions to them.

The word "social" is not to be confused with "socialized" in discussing the subject of medical economics. First, let us define "medical economics." Economics is the study of the means by which people obtain the goods and services they need or desire. By adding the word "medical" to "economics" we refer to the means by which people obtain the personal services of physicians, dentists, and nurses, hospitalization, and medicines which the present stage of scientific knowledge affords for the diagnosis and treatment of disease or injury. "Health economics" is something else again. Here we are concerned not only with medical services, but with the matter of sanitation, epidemiology, research, housing, nutrition, and social psychology—all those things which contribute to or detract from the health of the individual and the social group.

"Definitions are hazardous," said Samuel Johnson. Nevertheless, I think we may profit by considering, at the outset, the definition of the words "social" and "socialized" as they pertain to medicine. They are words of high abstraction and mean different things to different people.

A few years ago, in an effort toward elucidation, I wrote a letter to a large number of persons prominent in the controversy over compulsory sickness insurance, both in this country and in England. I asked them to give me their definition of some of the

words commonly used in discussions on the subject, as, for instance, "social medicine," "socialized medicine," "state medicine," "government medicine," "nationalized medicine," "political medicine," and "compulsory sickness insurance." The response was gratifying, but the result was dismally disappointing. No one was wholly satisfied with Webster's definitions, and no two people attached the same connotation to a single one of these words.

The word "socialism" was coined in England in 1835 to describe the theories of social reconstruction advocated by Robert Owens. It would seem appropriate, therefore, to turn to England for the definitions of the words "social" and "socialized." Significantly, it seems to me that the most satisfactory definitions I obtained in my search for lucidity came from Lord Horder, a leading figure in British medicine, and I think I am justified in quoting him at some length. His style is engaging and his ideas on the subject are manifestly the result of more than cursory meditation. He wrote (1):

"By nationalizing medicine, I mean doing with medicine what the Socialist desires to do with the land, the banks, the coal mines, and the railways—bring them under the control of the State. This is equivalent to a whole-time State service for all doctors, and that is what I mean by 'nationalized medicine.'

"The term 'State medicine' is not free from ambiguity, for we have a State Medical Service already—a service which is certain to extend after the war. 'Socialized medicine'—another term that is being used quite often—is more ambiguous still. Socialists use the term 'State medicine' and 'socialized medicine' as synonymous. They mean by both that the State takes control of the medical profession in all its branches and in all the media through which it operates.

"Then there is confusion as between 'socialized medicine' and the new term 'social medicine.' Since Professor Ryle occupies the first chair in social medicine, and is its chief apostle, we may accept his

definition without demur. Social medicine, he says, is concerned 'with the many and varied problems created by sickness in the family and the community as a whole.... It embodies the idea of medicine applied to the service of man as *socius*, as fellow or comrade, with a view to a better understanding and more durable assistance of all his main and contributory troubles which are inimical to active health and not merely to removing or alleviating a present pathology. It embodies also the idea of medicine applied in the service of *societas*, or the community of men, with a view to lowering the incidence of preventable disease and raising the general level of human fitness.'

" 'Social medicine' is not a new concept in medicine but is a timely and justifiable challenge, both to medicine and to the State, that they have left undone those things which they ought to have done. But such is my view; for it may readily be conceded that, in searching for the aetiology of disease, medicine has not probed deeply enough into the lives and conditions of the people, so as to bring to light the more ultimate factors that cause unfitness, sub-health, and actual disease. So also it may be readily conceded that in treatment, and especially in preventive treatment, dictated by the discovery of these ultimate factors, medicine has not been nearly as vocal as it might have been. If, for example, overcrowding is a determining factor in the causation of rheumatic fever, then it was the business of medicine to discover this fact, to take it into account when dealing with the indications for preventive treatment, and so point out the position to those whose duty it was to take action. On the other side, our statesmen, who are at the moment paying so much lip service to what is called 'positive' health, have so far done little to ensure for the people the basic conditions that make health possible.

"What are these basic conditions? First, enough of the right food; for to preach fitness to undernourished is sheer hypocrisy. Second, shelter, but shelter at a rent which leaves something to buy food with and pay for transport to and from work. Third, easy access to the fresh air and the sun. Fourth, leisure for play; and—who knows—that may lead to thinking, even the 'high thinking' which the poet lamented is 'no more.' Fifth, the amenities, amongst which you will not expect me to omit noise control. [Lord Horder, as you know, has acquired some reputation for his belief that one of the worst abominations of modern civilization is the blatant noise which accompanies it.] And last, the giving to every human being a chance before he is born and, after death, the decent disposal of his body in the best interest of his fellowmen."

It is said that most disputation comes from the want of accurate definitions. Accordingly, I submit the following peremptory definitions for the purposes of our

present discussion. (I differ somewhat from Lord Horder in my definitions, from those appearing in the *Encyclopedia Britannica*, and from those adopted by the American Medical Association—but that is my right.)

*Social medicine* is that aspect of medical diagnosis, treatment, palliative care, or preventive medicine that pertains to or affects society as a community of aggregate persons. It is a factor in "health economics."

*Socialized medicine* connotes a system of medical practice in which the method of the distribution of medical care is made subject to the uses or influences of a political agency, such as the state.

Socialized medicine is one method by which one of the aims of social medicine has been sought. All systems of socialized medicine are, in their objectives, technics of social medicine. The reverse is not true.

My definitions may be illustrated with examples. Russia's system of *state* medicine is socialized medicine. So also is Britain's system of *nationalized* medicine. Medical care in the armed forces of the United States and the Veterans Administration is socialized medicine.

State laws controlling the right to practise medicine are social laws, but they have nothing to do with socialized medicine because they do not pertain to the *distribution* of medical care. Health and accident insurance, voluntary sickness insurance, and Blue Cross hospitalization insurance are concerned with the distribution of medical and hospital care, but they are not socialized because they are not subject to the control of government. They are developments in social medicine.

Now, in this symposium on socialized medicine, I have been assigned the subject, "The Implications of Social Medicine for Radiologists." Actually, of course, one cannot discuss *social* medicine in the present state of affairs without also discussing *socialized* medicine, because socialized medicine is being advanced by many zealous advocates as the only means of achieving the aims of social medicine.

As a member of the medical profession, the radiologist is naturally concerned with all of the many and diverse aspects of social medicine. If, for example, the radiologist can reduce the death rate from heart disease and cancer, or if he can help to eradicate tuberculosis, he has made a contribution of enormous social implications. It is estimated that 4,391,000 potential years of productivity are lost each year in the United States through premature deaths from these three diseases (2). Thus disease acquires social significance.

But the *science* of medicine is not a matter of controversy. Since 1830, when Pierre Louis developed the first clinical statistics by having one large group of pneumonia patients bled while a control group was not bled, the astonishing progress in the knowledge and skill of the profession has received universal acclaim. The controversy arises over the question of what is the best and most effective method for the distribution of the services the medical profession is equipped to render—in short, how to pay for them.

Since the beginning of the century there has been a growing demand in this country that more adequate medical care be made available to more of the people. This poses a problem. As the magazine *Fortune* has stated: "The state of medicine in the United States is a social problem because the country's conscience has made it so—people who cannot find or pay for medical care are resentful."

Much has been written and said of the lack of doctors, nurses, and hospitals in certain areas, of the inability of the poor to pay for medical service, and of the failure of the medical profession to adjust itself to social needs. But there is no general agreement as to the nature, extent, and urgency of these needs. It is stated that the United States is the healthiest of nations and, conversely, that we lag behind other progressive lands in this respect and that therefore a revolution in the methods of providing medical care is imperative. There is debate as to the form this change

should take. Some argue for voluntary insurance against the costs of sickness; others for a compulsory tax collected by the government, the funds thus acquired to be used for paying for medical care received by all the people. These alternatives involve the basic questions of free initiative *versus* government planning—questions on which the American people are divided.

After thirty-five years of limited socialized medicine, the people of England will next year completely socialize medicine, just as they have the mines, in what Lord Horder refers to as a system of nationalized medicine. Do the people of the United States want this? I think not.

On Sept. 18, 1947, the socialist government of Great Britain issued a directive which compels workers to accept work to which they are assigned by the government. Persons engaged in certain vocations are forbidden to change to another. A worker applying to a government employment exchange who refuses to take work to which he is assigned by the government is subject to a fine of \$400 or three months in prison or both. Not since 442 A.D., when the Roman legions withdrew from England, has a British freeman been told where and when he must work. Have we then witnessed a complete cycle in political government since the English barons compelled King John to put his signature on the Magna Charta at Runnymede?

Here before our very eyes we have a glaring example of the postulate so convincingly presented by Ludwig von Mises: when the government begins to intervene in the lives of its citizens, there is no turning back (3). Just a little intervention is too much; it leads inexorably to complete socialism and the police state. Bureaucracy piles upon top of bureaucracy until the individual is engulfed in the meshes of a total state. What England is suffering today is not a crisis of capitalism, it is a crisis of interventionism, of a planned economy that failed to work. Now to cure the evils inherent in it, they impose more interventionism. We have seen that there is no such thing as

"mixed economy"—basic capitalism with just a little socialism here and there. Either we must have capitalism with a free market economy allowing each individual to work and earn his own degree of security, or we shall have socialism with security handed down by a paternalistic state in return for individual freedom. It appears that there is no middle way.

I am reminded of the opening statement in an address Mr. Walter Linn was recently invited to present under the title "Dangers to Our American Way of Life." This, he said, was as though he had been invited to speak on "Dangers to the Southern Confederacy," or "How to Preserve the Bison Herds on Our Western Plains" (4).

"There is no Southern Confederacy," he said. "There are no bison herds, and there is no American Way of Life. Only the more mature among you can even remember when there was an American way of life, as distinct from the European way of life. Little by little it has been chiseled away, much as Lord Macauley nearly ninety years ago prophesied that it would be.

"Historians may differ as to just when this process of decomposition began, but those of us who were old enough to observe can realize now that it was plainly perceptible prior to the first World War, and that it had been tremendously accelerated long prior to the second World War, which put a capstone on it. It was when the United States Supreme Court declared the Social Security Law constitutional on May 24, 1937, that I first became convinced that the American way of life was ended beyond resuscitation in our day and generation."

Now, whether the disease of socialism has already sapped the vitality of free America, whether the Social Security Act was only the beginning act of interventionism that will lead inexorably to more and more manifestations of collectivism, whether the "general welfare" clause in the constitution is to be used by the socialists to impose the theories of Karl Marx upon the people of America—this is precisely the question presented in the proposals for a Federal sickness tax to be found in the Wagner-Murray-Dingell bill. It is not too much to say that the decision on this legislative proposal will determine the system of political economy under which the

citizens of this country will live in the future. These are the years of decision.

It was exactly one hundred years ago this month that the Communist League, at its congress in London, commissioned Karl Marx and Frederick Engels to prepare a manifesto setting forth the views, aims, and principles of the Communists. In the document they produced they perpetrated a colossal myth that has been preserved during the century that followed. I don't know how the Germans would have expressed it in 1847, but in the year 1947 in the United States of America we would say that Karl Marx missed the boat. The myth he perpetrated is that society as a whole is more and more splitting up into two great hostile camps, into two great classes facing each other in conflict—bourgeoisie and proletariat.

A century ago the Communists saw the middle class disappearing. They envisioned a small group of capitalists owning all the sources of production and employing the proletariat as slaves like the serfs of feudal lords. The proletariat would become a class of wage slaves who, having no means of production of their own, would be reduced to selling their labor to capitalist barons at a price which would place the great masses of humanity at an economic and social level of want, poverty, and misery.

Quite the contrary has occurred. The middle class has not disappeared; the poor have practically done so. The working man in modern capitalistic America does not wallow in miserable poverty. He wears white shirts, drives a shiny new automobile, has a radio in one or more rooms of his house, and his wife purchases one dollar pork chops at the corner market while affecting the "new look" as handed down by the fashion decrees of Hattie Carnegie.

To be sure, there is poverty in America. But the poor today are victims of circumstances, they are not victims of a system, or of the predatory greed of capitalists who deny them a fair share of this world's goods. The rich man in America today works for his living the same as the poor

man, and the hiatus between the rich and the poor seems constantly to be narrowing. The fifty largest corporations in this country are managed by 143 top executives. A recent study revealed that every one of them started at the bottom and came up through the ranks. What we have witnessed here is not a communist revolution of the proletariat in a bloody struggle with the bourgeoisie. Rather, it has been a managerial revolution with those who can accomplish things occupying the best places in our society. The doom of the idle rich was sealed with the enactment of the sixteenth amendment to our constitution providing for a tax on incomes. Ours is today approaching a classless society. Agitation for socialist measures in our country comes not so much from the workers as from the "intellectuals"—the seekers after Utopia—just as was the case in England.

It is true, of course, that there are ambitious Caesars in our government who want to socialize medicine and impose the doctrines of totalitarian Europe on our people because it will advance their political fortune. I have not yet forgotten the awful implications in the message the late Senator J. Hamilton Lewis came to deliver for the administration before the American Medical Association in 1937 in Atlantic City. He said this: "We know nothing of a patient, don't recognize his existence. We recognize an instrument called a citizen who is essential to the welfare of government."

That, in the land of the free, and the home of the brave!

Neither should we overlook the fact that socialized medicine was established in Germany in 1883 and by David Lloyd George in England in 1911 as a matter of political expediency.

But we would delude ourselves if we contended that all the visionary reformers who advocate compulsory sickness insurance were venal in their motives. There are some honest, reasonable men who sincerely believe that compulsion is the only means of achieving the ends of social medicine. We may take a leaf from the first number

of *The Federalist* in which Hamilton wrote: "Candor will oblige us to admit that even such men may be actuated by upright intentions; and it cannot be doubted that much of the opposition which has made its appearance, or may hereafter make its appearance, will spring from sources, blameless at least, if not respectable—the honest errors of minds led astray by preconceived jealousies and fears. So numerous indeed and so powerful are the causes which serve to give a false bias to the judgment, that we, upon many occasions, see wise and good men on the wrong as well as on the right side of questions of the first magnitude to society. This circumstance, if duly attended to, would furnish a lesson of moderation to those who are ever so much persuaded of their being in the right in any controversy. And a further reason for caution, in this respect, might be drawn from the reflection that we are not always sure that those who advocate the truth are influenced by purer principles than their antagonists (5)."

It is medicine's job to convince the majority of the citizens, by word and deed, that they would lose much more than they would gain if medical care were furnished for all the people by means of a tax levied on their payrolls.

But, it is said, even the middle class is unable to meet the cost of sickness. A report emanating from the American Medical Association is quoted and interpreted as admitting that families with incomes of \$3,000 or under are unable, at least at times, to pay the costs of sickness. This may very well be true. But the persuasiveness and the pathos of such a statement lose some of their appeal when we note that in 1946 the public spent a greater proportion of its income for liquor than it did for medical care. Last year the American people spent 3.3 per cent of their income for medical care. This amounted to \$5,600,000,000. In the same year they spent 5.2 per cent of their income, totaling \$8,800,000,000 for alcoholic beverages, 2 per cent or three and one-half million dollars for tobacco, and 4.7 per cent or

nearly eight billion dollars for recreation (6). It looks very much as though the American people can afford to pay for all the medical care they need under a system of free enterprise and private practice.

It would be idle to deny that there are occasions, created by long or serious illness, when even the well-to-do find it difficult to meet the cost of medical care. Neither can one deny that there are people, even in these times of frenzied prosperity, in such a state of penury that they cannot afford to pay for even minor medical services. But this presents no valid reason for revolutionizing our system of political economy by a sickness tax on all the people as proposed in the Wagner-Murray-Dingell bill. We have perfectly adequate answers to both these problems in our present social and economic systems.

For the first, we have voluntary insurance that will spread the cost of serious illness over a large group of consumers and over a period of time. As of the first of this year, there were 39,690,000 persons insured against the costs of hospitalization. Of these, 16,064,000 were covered for the costs of surgery and obstetrical care. Almost 5,000,000 were covered by insurance against any type of illness serious enough to require hospitalization. And 3,666,000 enjoyed complete protection against all hospital and medical bills, including services in the home or the physician's office (7). Some 20,000,000 persons, including, of course, many of those already counted above, were entitled to workmen's compensation benefits, and millions own personal health and accident insurance policies (8).

These figures become even more convincing when we realize that the voluntary sickness insurance movement actually began only about seven years ago. People who want to can today protect themselves against the unpredictable costs of illness through methods available to them without taxation, interventionism, or bureaucracy.

For the second group, those too poor to pay for even minor illness and unable to

afford premiums for voluntary insurance, our present social system also provides an answer. What these people need, and what they receive, is charity. In every city in America there exist eleemosynary agencies through which adequate medical care of high quality may be obtained by those too poor to pay for it.

In rural areas, particularly in the south, the problem is not so simple. More than half of all farm families had cash incomes of less than \$1,000 in 1941, and a third had incomes under \$500 (9). Obviously these people cannot translate into effective demand their needs for medical care. But poverty is only part of the problem in rural areas. It is complicated by such factors as isolation and ignorance.

Dr. Maurice H. Friedman, in testimony submitted to the Senate Committee on Labor and Public Welfare on June 25, 1947, endeavored to show (not very convincingly, I think) that low-income families in rural areas have less sickness than prosperous families. Dr. Friedman presented an eloquent argument against socialized medicine, but a statement of this nature simply cannot be substantiated. Common observation, to say nothing of a number of statistical surveys, demonstrates that there is a definite lack of adequate medical care in many of our poor rural areas.

But this would seem to be insufficient reason to institute a system of nationalized medicine for all the people. Furthermore, the passage of the Wagner-Murray-Dingell bill would not of itself be a guarantee that adequate medical care would become available to families living many miles from a town in sparsely populated and substandard agricultural areas. Neither poverty nor lack of medical facilities is alone responsible for the fact, for example, that the death rate from pneumonia in Kentucky is 89 per 100,000, while in the District of Columbia it is only 42 per 100,000. Ignorance, superstition, poor housing, and nutritional deficiencies must accept a share of the blame. These are social problems; they are not medical problems.

Partly to meet this problem, Senator

Robert A. Taft has introduced a bill to supply grants-in-aid to states to assist in furnishing medical services to families unable to pay the full cost of such care. You are all familiar, I am sure, with the provisions of Senate Bill 545. I confess that I have misgivings about the Taft bill, but, if it is necessary that the Federal Government appropriate tax funds for medical care—a matter concerning which I am not entirely persuaded—then the Taft bill represents a sound and intelligent method for the accomplishment of the objective sought.

I know I shall be accused of the most outmoded kind of anti-social Toryism, but I wonder if it would not be better to allow the normal forces of a free market economy to solve the problem of medical care for substandard rural areas. If left alone, the people in these unfavored regions would eventually leave a locality where they cannot earn enough to provide themselves with the necessities of life and move to a region where the soil is kinder or where, consistent with the flow of labor in a free economy, they can earn a decent living. As it stands, no magic of legislation will make a medical graduate eager to establish himself in an area where his patients are many miles apart and where there are no decent schools for his children.

Here, of course, is one aspect of social medicine with important implications for radiology. Like Lord Horder, I think medicine has frequently been laggard about meeting the social responsibilities that inhere in the political freedom American medicine has heretofore enjoyed. We are opposed to the socialization of medicine. Radiologists are particularly opposed, and rightly, to the theory advanced by certain people in government that only through the establishment of tax-supported free diagnostic centers can adequate diagnostic services be made available to certain segments of our population.

But what have the radiologists done about this problem? Granting that no economic legerdemain will overcome the fact that every town and hamlet cannot

support a competent radiologist, has any attempt been made even to improve the situation? One should not be surprised, I suppose, that the distribution of radiologists in the United States is influenced almost entirely by variations in the average per capita income of the population. But the fact remains that the residents of poorer, more sparsely settled areas want and need radiological services which are not now available to them.

According to the Lee-Jones study, based upon morbidity statistics, 228 x-ray examinations per 1,000 population would be required for "adequate" medical care (10). Others have estimated that one radiologist can render the minimum diagnostic procedures required by about 50,000 people. On this basis, New York, with the highest per capita income, has 102 more radiologists than are needed to adequately serve the population. Its minimum need is 252; there are 354 practising in the state. Mississippi, at the other end of the income scale, needs 42 radiologists, and has only 4 (11).

Each year the science of radiology becomes more essential in the armamentarium of modern science. As its value in more and more disease conditions increases, the number of people a single radiologist can adequately serve will be constantly decreased. This presents a challenge for the profession of radiology. Unless radiological facilities can be made widely available under a system of free enterprise, then we may be compelled to yield to a solution offered by government. As always, free enterprise must adapt itself to the exigencies of society if it is to survive.

Here again we are confronted with the basic questions of controversy I referred to in the beginning; what is the best and most effective method for the distribution of medical services and how should they be paid for? This is a paramount question for all medicine in America today, but for radiology it is complicated by one vital development in the economics of medicine that has heretofore been largely disregarded by organized medicine. I refer, of course,

to the insidious and unmistakable tendency for hospitals to become distributing agencies for medical care through the medium of employed physicians.

Whether we like it or not, the fact is that a considerable proportion of the total service rendered by radiologists is rendered in hospitals. Perhaps this is an inevitable and inescapable result of technical advancements in medical practice and the gradual urbanization of our population. At any rate, about 50 per cent of all serious illnesses are today treated in hospitals, and the proportion seems to be increasing. Obviously, the relationship between the radiologist and the hospital becomes extremely important. It becomes doubly important when we consider the rapid growth of medical insurance. How will this development in social medicine affect radiology?

If radiology is practised like any other specialty in the hospital, then we have no special problems peculiar to this branch of medicine. If, on the other hand, the hospital becomes a third-party, intermediary in the delivery of radiological services, then our problem is greatly complicated. The unpleasant fact is that in many hospitals radiology is treated differently from medicine or surgery, for instance. Too often the radiologist is in the legal position of an employee of the hospital.

Some years ago, in a study conducted by the American College of Radiology, we found that 37 per cent of all radiologists were salaried employees of hospitals. Only 9 per cent leased the department at a fixed rental just as they would a private office, or paid the hospital a fee per case. The remaining 54 per cent were in the legal position of employees on a commission basis.

Now, it is a waste of time to say that radiologists themselves are partly responsible for this situation. Everybody knows that. What we must do is improve the situation—and I am happy to say that there are encouraging evidences of improvement. Every year there are more and more radiologists who succeed in changing their position from that of an employee on

a commission basis to that of a tenant, renting the x-ray department as they would a private office and paying a fixed rental or a rental based upon a percentage of gross or net income. Too, an increasing number are collecting their fees in their own department, or allowing the hospital to collect them on their own billheads.

There is reason to be encouraged too, I believe, by increasing evidences that the general medical profession is slowly beginning to recognize the truth of our warning that, if hospital corporations can practise radiology through the medium of salaried agents, they can also practise obstetrics, and surgery, and general medicine. I could quote innumerable instances of this manifestation of a growing awareness on the part of the general profession.

I sincerely believe we have reason to be reassured and encouraged in this connection. True, our problems are not going to be corrected overnight. This situation developed over a period of thirty years, and we are not going to succeed in correcting it in a year or two. We will simply have to keep working at it relentlessly and patiently.

I hope, if you are not already fully aware of it, that I can convince you of the tremendous importance of this principle. If 80 per cent of all radiologists collected their own fees in the hospital instead of *vice versa*, as is the case, and if the majority of radiologists were tenants instead of employees of the hospital, our problems would be precisely those of the rest of medicine. We would have no controversy with Blue Cross. In half the Blue Cross plans throughout this country radiology is regarded as a medical service and is not included among the benefits which hospitals guarantee to furnish subscribers. In the other half of the plans, including those in some of our largest cities, we have been unable to overcome successfully the argument that Blue Cross is insurance for hospital care and that most people regard radiology as a part of hospital care.

The Wagner-Murray-Dingell bill contemplates that x-ray services shall be furnished

by hospitals and included in the *per diem* they receive for hospitalization. This never would have happened if dangerous precedents in the relationship between radiologists and hospitals had not been established years ago.

The Veterans Administration is currently effecting contracts with state medical societies which permit veterans with service-connected disabilities to select the physician of their choice who will be paid on an agreed, and very fair, fee schedule by the Veterans Administration. These contracts include radiology as a part of medical care and provide a fee schedule for x-ray services, but in one state at least the medical society permitted radiology to be included in hospital service and the Veterans Administration pays hospitals on an inclusive *per diem* for radiology, pathology, and anesthesiology.

The growth of voluntary medical service plans sponsored by medical societies presents both a challenge and an opportunity to radiology. We must see to it that radiology is included among the benefits offered by these medical or surgical plans, instead of being regarded as a part of hospital care. This may be the very best means at our disposal for freeing the radiologist from the domination of the hospital.

I cannot emphasize too strongly the enormous importance of achieving for radiology a proper role in the development of these voluntary medical insurance plans. There are some reasonable men in this country, including a recent past president of the American Medical Association, who believe that compulsory sickness insurance will come in the natural course of events; if not now, then later. Personally, I think they are wrong, but on the bare chance that they are right, there is one profoundly important point to be kept clearly in mind. It is this: Every system of compulsory health insurance in all the countries of the world has been built upon existing agencies for the distribution of medical care. On the basis of history, therefore, we can assume that, if a system of compulsory health insurance is adopted in this country,

existing plans for the application of the insurance principle to the payment for medical care would be utilized by the state. The obvious corollary is that medical practitioners would carry on under the state plan much as they did under the voluntary plans which preceded it. This has been almost the universal experience in European systems. Indeed, there are provisions in the Wagner-Murray-Dingell bill which contemplate the incorporation of these voluntary plans in the national health service.

Another fact should be emphasized. Sociology is not a science for the same reason that medicine is. You can follow a certain theory or employ a certain technic in the treatment of cancer for five or ten or twenty years. Then, after you have treated many thousands of patients, you can, if you wish, one day undertake an entirely different approach to cancer. You can start out fresh; what you did before does not affect the results you will obtain on new patients now. But, in social experimentation, you can never escape past errors. You can never make a fresh start. As my friend, A. M. Simons, has said, social experiments invariably establish patterns of precedent that are seldom completely erased.

These facts offer sufficient evidence, I believe, of the impelling necessity of correcting the dangerous precedents already allowed to develop in the practice of radiology in hospitals, and the urgent importance of achieving a proper role for radiology in voluntary medical and hospital insurance. Each individual radiologist has a responsibility in this regard. We can't expect the impossible; no one has suggested that every radiologist who is employed on a salary by a private general hospital should resign tomorrow. But we can continue to emphasize the principle involved, we can continue to warn the general profession that the integrity of all medicine is at stake; we can encourage young radiologists beginning practice to insist upon an arrangement with their hospital under which they will remain independent private practitioners and

not become employed agents of hospital corporations.

This, you will observe, is the program of the American College of Radiology. The social and economic forces of America are articulated through strongly organized minorities, many of them with enormous funds at their disposal. The College, representing the combined and unified voice of all radiology, is making its force felt in influencing the direction of social, economic, and political developments. By formulating and articulating principles of good radiological practice, it is endeavoring to preserve, amid the complexities of a dynamic society, the magnificent contributions already achieved and yet to be accomplished by radiology for human health and welfare.

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#### REFERENCES

1. HORDER: Shall We Nationalize Medicine? *Brit. M. J.* 1: 357-360, March 17, 1945.
2. DEMPSEY, MARY: Decline in Tuberculosis; Death Rate Fails to Tell the Entire Story. *Am. Rev. Tuberc.* 56: 157-164, August 1947.
3. VON MISES, LUDWIG: *Planned Chaos*. Foundation for Economic Education, Irvington-on-the-Hudson, 1947.
4. *Vital Speeches of the Day*, March 15, 1947.
5. HAMILTON, ALEXANDER: *The Federalist* (Number 1). New York, The Heritage Press, 1945, p. 2.
6. *Survey of Current Business*: Bureau of Foreign and Domestic Commerce, U. S. Department of Commerce, Washington, D. C., July 1947.
7. REED, LOUIS S.: *Blue Cross and Medical Service Plans*. Federal Security Agency, Washington, 1947.
8. *Toward Better Health*. Research Council for Economic Security, Chicago, 1947.
9. *What Farm Families Spend for Medical Care*. U. S. Department of Agriculture, Miscellaneous Publication No. 561, Government Printing Office, Washington, D. C., April 1945.
10. LEE ROGER I., AND JONES, LEWIS W.: *The Fundamentals of Good Medical Care*. Chicago, University of Chicago Press, 1933.
11. DAVIS, GRAHAM L.: Wanted: 381 Pathologists, 819 Radiologists. *Hospitals* 21: 49, 1947.

#### SUMARIO

##### **Certamen sobre la Medicina Socializada Celebrado en la Trigésimatercera Reunión Anual de la Sociedad Radiológica de Norte América**

Expónense los peligros inherentes en la "socialización," o hablando con mayor exactitud, control por el Gobierno Federal del ejercicio de la medicina; señálanse las erradas premisas en que se basa la legislación que busca dicho control, y se ponen en claro las connotaciones políticas. Se advierte a los radiólogos que estén en guardia contra proyectos de ley pendientes que se proponen legalizar la intervención del gobierno en la medicina.

# EDITORIAL

## Erythema Nodosum

There are numerous diseases in which the roentgen manifestations have long been observed and the clinical picture is well documented but the exact etiologic background has remained obscure in spite of repeated studies contributing to its determination. Such a disease is erythema nodosum. The pulmonary and lymph node changes have long been recognized roentgenographically and the clinical manifestations are well known to dermatologists. There has never been any real agreement, however, regarding the underlying cause. Various writers have stressed an association with other diseases, as observed in their experience, and in recent years the role of hypersensitivity has received considerable attention.

During the earlier decades of this century, erythema nodosum was generally believed to be an unusual manifestation of tuberculosis, and according to Favour and Sosman (1) that view is still the prevailing one in Great Britain and Scandinavia, though tubercle bacilli are notably absent from the lesions. In many cases, however, clinical tuberculosis has been present in some form. Paul and Pohle (3) studied a series of 20 cases of erythema nodosum for which chest roentgenograms were available and in 12 of these observed varying degrees of mediastinal or pulmonary change. In 6 of the series there was a moderate to pronounced hilar lymphadenopathy with evidence of tuberculosis which led the authors to regard that disease as the cause of both the erythema nodosum and the lymph node involvement.

In a roentgen study of the chests of 37 adults, Kerley (2) found evidence of recent disease in 28: 17 had enlarged bronchial nodes with pulmonary changes, 8 had

enlarged bronchial nodes without pulmonary changes, and 3 had pulmonary disease without node enlargement. The enlarged nodes were for the most part bilateral, while the pulmonary infiltration in half the affected cases was unilateral. The node enlargement was prominent and presented no difficulty in diagnosis except for its similarity to lymphomatoid disease. The pulmonary lesions were of several types. The commonest consisted in coarse reticular striations radiating from both hila, denser and sharper in outline than blood vessels and extending, undiminished in size, to the peripheral lung fields. In addition, there were pin-head spots similar to the nodulation of silicosis or lymphangitis carcinomatosa, probably due to lymphatic obstruction. In another type there were round or oval foci varying from 3 to 5 mm. in diameter. These were located either in a single lobe or were scattered throughout both lungs. Occasionally they coalesced to form areas of opacity 2 or 3 cm. in diameter. In some instances there were combinations of linear striae and pulmonary nodules, simulating silicosis.

Favour and Sosman, in a recent comprehensive review of erythema nodosum, preface the account of their own observations in 155 cases by a critical review of the literature. In addition to tuberculosis, they mention as associated conditions to which a causative role has been attributed, streptococcal infection, especially of the upper respiratory tract, and rheumatic fever. In 55 of their own series of cases cultures were made from material from the throat, and in half of these beta hemolytic streptococci were grown. These writers do not believe that erythema nodosum is a form of rheumatic fever, in spite of the

fact that 11 per cent of the patients have been said to have had pre-existing rheumatic heart disease, that migratory polyarthritis may be present in both conditions, and that rheumatic heart murmurs have been observed. Admitting that erythema nodosum and rheumatic fever may occur together, they still regard such concurrence as unusual and coincidental. Nor do they regard erythema nodosum as a form of sarcoid, since the clinical course of the two conditions shows no conformity. The frequency of erythema nodosum in association with coccidioidomycosis is also mentioned by these writers (5 per cent of 8,000 cases according to Smith), as well as its occasional occurrence in syphilis.

As to their own series of cases, Favour and Sosman obtained roentgenograms of the chest during the active stage of the disease in 65 patients. In 37 the findings were entirely normal in all respects, in 18 the pulmonary infiltration was non-specific, consisting in 13 cases of a slight accentuation of the normal lung markings, in 2 instances of a diffuse bilateral mottling similar to that seen in the earliest stages of silicosis, and in 3 of small areas of localized apical infiltration. In only 10 patients were there enlarged lymph nodes either at the roots of the lungs or in the upper mediastinum or both. In 6 of these patients the enlargement was limited to the bronchial nodes and was bilateral and fairly symmetric. In 3 there was a definite clear zone between the shadows and the mediastinal shadow of the heart and great vessels, a finding which the authors be-

lieve is the only pathognomonic evidence by which the enlarged nodes of erythema nodosum can be differentiated from other forms of lymphadenopathy.

We have presented at some length the findings of Favour and Sosman because of their bearing on the controversial aspects of erythema nodosum. It is evident that the etiology is not always apparent and that the disease may follow or accompany various toxic conditions. It is clearly established that tuberculosis is not the specific cause but that a variety of other bacterial and even chemical toxins may provoke the classical signs and symptoms. Wallgren (4) has stated that erythema nodosum should be regarded as a non-specific allergic cutaneous eruption which appears especially in tuberculosis but occasionally in other infections or may even possibly be caused by a non-infectious agent. Favour and Sosman regard it as a hypersensitivity disease, individual predisposition, a variety of infections, chemical agents, and local trauma contributing to its occurrence.

#### REFERENCES

1. FAVOUR, C. B., AND SOSMAN, M. C.: Erythema Nodosum. *Arch. Int. Med.* 80: 435-453, October 1947.
2. KERLEY, PETER: The Significance of the Radiological Manifestations of Erythema Nodosum. *Brit. J. Radiol.* 15: 155-165, June 1942; Etiology of Erythema Nodosum. *Brit. J. Radiol.* 16: 199-204, July 1943.
3. PAUL, L. W., AND POHLE, E. A.: Mediastinal and Pulmonary Changes in Erythema Nodosum. *Radiology* 37: 131-137, August 1941.
4. WALLGREN, A.: Erythema Nodosum and Pulmonary Tuberculosis. *Lancet* 1: 359-363, Feb. 12, 1938.

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## ANNOUNCEMENTS AND BOOK REVIEWS

### OAK RIDGE INSTITUTE OF NUCLEAR STUDIES

Announcement is made of a series of three one-month courses to be held at Oak Ridge in the technique of using radioisotopes. The courses will be conducted by the Oak Ridge Institute of Nuclear Studies, from June 28 to July 23, Aug. 2 to Aug. 27, and Aug. 30 to Sept. 24, 1948. Application forms and additional information may be obtained from Dr. Ralph T. Overman, Acting Head of the Institute's Department of Special Training, P. O. Box 117, Oak Ridge, Tenn.

These courses will not attempt to cover any special field of application in chemistry or biology but will be based on simple chemical experiments selected and designed to give participants a maximum knowledge and ability in the techniques of handling and carrying on research with radioisotopes. Participants in the second of the three courses will be selected by the Atomic Energy Commission. Personnel of the other courses will be chosen from qualified applicants, with preference given to persons connected with organizations which are now engaging in research utilizing radioisotopes or planning such research. Thirty-two participants will be selected for each course.

A fee of \$25.00 will be charged for the course. Participants will be expected to pay their own travel and living expenses. Dormitory or hotel accommodations will be provided at the usual rates.

### Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

**TECHNIQUE D'IRRADIATION DES TUMEURS MALIGNES. ROENTGENTHERAPIE-CURIETHERAPIE.** By CH. GUILBERT, Ancien chef de service radiologique à l'Hôpital Lariboisière. A volume of 300 pages, with 45 figures. Published by G. Doin & Cie, Paris 6<sup>e</sup>, 1947. Price 650 fr.

**DIAGNOSTIC ET TRAITEMENT DES MALADIES DE LA COLONNE VERTÉBRALE.** By JEAN SAIDMAN. Two volumes of 1,244 pages, with 1,324 illustrations. Published by G. Doin & Cie, Paris 6<sup>e</sup>. In one volume 4,500 fr.; in two volumes 4,750 fr.

**ANATOMIE RADIOLOGIQUE NORMALE. OPTIQUE RADIOLOGIQUE ET DÉPISTAGE DES ERREURS DE LECTURE DES CLICHÉS.** By HENRY TILLIER, Électro-radiologiste des Hôpitaux d'Alger. A volume

of 234 pages, with 350 figures. Published by G. Doin & Cie, Paris 6<sup>e</sup>. Price 600 fr.

### Book Reviews

**CONGENITAL MALFORMATIONS OF THE HEART.** By HELEN B. TAUSSIG, M.D., Associate Professor of Pediatrics, Johns Hopkins University School of Medicine, and Director of the Children's Cardiac Clinic at the Harriet Lane Home of the Johns Hopkins Hospital. With a Foreword by EDWARDS A. PARK, M.D., Professor of Pediatrics, Johns Hopkins University School of Medicine. A volume of 572 pages with 223 illustrations and diagrams. Published by the Commonwealth Fund, New York City, 1947. Price \$10.00.

Dr. Taussig has written a fundamental monograph on a subject which is gaining in interest every day. As more surgeons become trained in the Blalock-Taussig operation, devised for relieving coarctation of the aorta and ligation of the patent ductus arteriosus, it will be the duty of radiologists everywhere to train themselves to pick out the cases suitable for surgery. For, as the author emphasizes throughout the work, the findings on fluoroscopy and radiography are the foundation of the diagnosis. The presence or absence of thrills means little or nothing in the individual case.

Complicated procedures are not necessary in the study of congenital heart disease. Catheterization of the heart chambers is seldom practical and almost never necessary. Angiocardiography, while of great theoretical interest, is of relatively little practical importance, since the information thus gained can be obtained by less spectacular (and safer) means. The author admits frankly that she has had no experience with it. Electrocardiography is used chiefly to determine the axis deviation and arrhythmias. It is the opinion of the reviewer that, with the aid of a few simple laboratory procedures, and careful analysis of the findings on fluoroscopy and radiography as outlined by Dr. Taussig, one should be able to diagnose most of the more common lesions and certainly to pick out those which might be advantageously treated by surgery.

The author first takes up the basic principles involved in the clinical study of the various malformations and gives a brief review of the embryology and physiology of the fetal circulation. The section on methods of diagnosis is almost wholly devoted to the techniques of fluoroscopic and radiographic examination. No complicated procedures or positions are used; just careful attention to the relative size of the various chambers and great vessels, the degree of vascularity of the lungs, the presence or absence of pulsations, etc

Cyanosis is thoroughly discussed in an interesting chapter, completing the background for the following sections. Then the various entities are taken up one by one, each being accompanied by clear and easily understandable diagrams of the anomaly and its effect upon the circulation. Each diagram is accompanied by a full page summary of the salient features of the condition.

The book concludes with chapters on medical and surgical treatment. A tabulated summary of the diagnostic points is appended which should be useful for quick reference.

In summary, the author has made an important contribution to medical literature in setting forth the results of her years of experience with these unfortunate patients, many of whom can now be enabled to lead fairly normal lives through surgery. The book is well written and it definitely belongs in the library of every radiologist.

LA RADIOTHERAPIE ANTI-INFLAMMATOIRE By JEAN HUGUET, Electro-radiologist des Hôpitaux de Marseille. A volume of 200 pages. Published by G. Doin & Cie, Paris 6<sup>e</sup>, 1947. Price 440 fr.

The author's aim evidently was not to prepare an exhaustive treatise, but rather to undertake a general review of roentgen treatment for inflammatory conditions, and this purpose has been largely fulfilled. While frequent reference is made to the work of others, a goodly part of the book is based on personal experience in dealing with various inflammatory lesions. Among the conditions which the author has neglected to consider adequately may be mentioned acute and chronic sinusitis and tuberculous lesions in general. This small book will be valuable to radiologists and to students who desire to keep abreast of this phase of therapeutic radiology; but it does not meet the need for a thoroughly satisfactory text on this subject.

Thirty-fourth Annual Meeting  
Radiological Society of North America  
Hotels Fairmont and Mark Hopkins  
San Francisco  
Dec. 5-10, 1948

## RADIOLOGICAL SOCIETIES: SECRETARIES AND MEETING DATES

*Editor's Note:* Secretaries of state and local radiological societies are requested to co-operate in keeping this section up-to-date by notifying the editor promptly of changes in officers and meeting dates.

### UNITED STATES

**RADIOLOGICAL SOCIETY OF NORTH AMERICA.** *Secretary-Treasurer*, Donald S. Childs, M.D., 607 Medical Arts Bldg., Syracuse 2, N. Y.

**AMERICAN RADIUM SOCIETY.** *Secretary*, Hugh F. Hare, M.D., 605 Commonwealth Ave., Boston 15, Mass.

**AMERICAN ROENTGEN RAY SOCIETY.** *Secretary*, Harold Dabney Kerr, M.D., Iowa City, Iowa.

**AMERICAN COLLEGE OF RADIOLOGY.** *Secretary*, Mac F. Cahal, 20 N. Wacker Dr., Chicago 6, Ill.

**SECTION ON RADIOLOGY, A. M. A.** *Secretary*, U. V. Portmann, M.D., Cleveland Clinic, Cleveland 6, Ohio.

### Alabama

**ALABAMA RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, Courtney S. Stickley, M.D., Bell Bldg., Montgomery. Next meeting with State Medical Association.

### Arkansas

**ARKANSAS RADIOLOGICAL SOCIETY.** *Secretary*, Fred Hames, M.D., Pine Bluff. Meets every three months and at meeting of State Medical Society.

### California

**CALIFORNIA MEDICAL ASSOCIATION, SECTION ON RADIOLOGY.** *Secretary*, Sydney F. Thomas, M.D., Palo Alto Clinic, Palo Alto.

**LOS ANGELES RADIOLOGICAL SOCIETY.** *Secretary*, Moris Horwitz, M.D., 2009 Wilshire Blvd., Los Angeles 5. Meets second Wednesday of each month at County Society Bldg.

**PACIFIC ROENTGEN SOCIETY.** *Secretary*, L. Henry Garland, M.D., 450 Sutter St., San Francisco 8. Meets annually with State Medical Association.

**SAN DIEGO ROENTGEN SOCIETY.** *Secretary*, R. F. Niehaus, M.D., 1831 Fourth Ave., San Diego. Meets first Wednesday of each month.

**X-RAY STUDY CLUB OF SAN FRANCISCO.** *Secretary*, Ivan J. Miller, M.D., 2000 Van Ness Ave. Meets monthly on the third Thursday at 7:45 P.M., January to June at Lane Hall, Stanford University Hospital, and July to December at Toland Hall, University of California Hospital.

### Colorado

**DENVER RADIOLOGICAL CLUB.** *Secretary*, Mark S. Donovan, M.D., 306 Majestic Bldg., Denver 2. Meets third Friday of each month, at the Colorado School of Medicine and Hospitals.

### Connecticut

**CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY.** *Secretary*, Robert M. Lowman, M.D., Grace-New Haven Hospital, Grace Unit, New Haven. Meetings bimonthly, second Thursday.

### District of Columbia

**RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY.** *Secretary*, Alfred A. J. Den, M.D., 1801 K St., N. W., Washington 6. Meets third Thursday of January, March, May, and October, at 8:00 P.M., in Medical Society Auditorium.

### Florida

**FLORIDA RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, J. A. Beals, M.D., St. Luke's Hospital, Jacksonville. Meets in April, preceding annual meeting of Florida Medical Society, and in November.

### Georgia

**GEORGIA RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, Robert Drane, M.D., De Renne Apartments, Savannah. Meets in November and at the annual meeting of State Medical Association.

### Illinois

**CHICAGO ROENTGEN SOCIETY.** *Secretary*, T. J. Wachowski, M.D., 310 Ellis Ave., Wheaton. Meets at the Palmer House, second Thursday of October, November, January, February, March, and April, at 8:00 P.M.

**ILLINOIS RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, William DeHollander, M.D., St. Johns' Hospital, Springfield. Meetings quarterly as announced.

**ILLINOIS STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY.** *Secretary*, John H. Gilmore, M.D., 720 N. Michigan Ave., Chicago 11.

### Indiana

**INDIANA ROENTGEN SOCIETY.** *Secretary-Treasurer*, J. A. Campbell, M.D., Indiana University Hospitals, Indianapolis 7. Annual meeting in May.

### Iowa

**IOWA X-RAY CLUB.** *Secretary*, Arthur W. Erskine, M.D., 326 Higley Building, Cedar Rapids. Meets during annual session of State Medical Society.

### Kentucky

**KENTUCKY RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, Sydney E. Johnson, M.D., 101 W. Chestnut St., Louisville.

**LOUISVILLE RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, Everett L. Pirkey, Louisville General Hospital, Louisville 2. Meets second Friday of each month at Louisville General Hospital.

### Louisiana

**LOUISIANA RADIOLOGICAL SOCIETY.** *Secretary-Treasurer*, Johnson R. Anderson, M.D., No. Louisiana Sanitarium, Shreveport. Meets with State Medical Society.

**ORLEANS PARISH RADIOLOGICAL SOCIETY.** *Secretary,* Joseph V. Schlosser, M.D., Charity Hospital of Louisiana, New Orleans 13. Meets first Tuesday of each month.

**SHREVEPORT RADIOLOGICAL CLUB.** *Secretary,* Oscar O. Jones, M.D., 2622 Greenwood Road. Meets monthly September to May, third Wednesday.

#### Maryland

**BALTIMORE CITY MEDICAL SOCIETY, RADIOLOGICAL SECTION.** *Secretary,* Harry A. Miller, 2452 Eutaw Place, Baltimore.

#### Michigan

**DETROIT X-RAY AND RADIUM SOCIETY.** *Secretary-Treasurer,* E. R. Witwer, M.D., Harper Hospital, Detroit 1. Meetings first Thursday of each month from October to May, at Wayne County Medical Society club rooms.

**MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS.** *Secretary-Treasurer,* R. B. MacDuff, M.D., 220 Genesee Bank Building, Flint 3.

#### Minnesota

**MINNESOTA RADIOLOGICAL SOCIETY.** *Secretary,* C. N. Borman, M.D., 802 Medical Arts Bldg., Minneapolis 2. Regular meetings in the Spring and Fall.

#### Missouri

**RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY.** *Secretary,* Wm. M. Kitchen, M.D., 1010 Rialto Building, Kansas City, 6, Mo. Meetings last Friday of each month.

**ST. LOUIS SOCIETY OF RADIOLOGISTS.** *Secretary,* Edwin C. Ernst, M.D., 100 Beaumont Medical Bldg. Meets on fourth Wednesday, October to May.

#### Nebraska

**NEBRASKA RADIOLOGICAL SOCIETY.** *Secretary-Treasurer,* Ralph C. Moore, M.D., Nebraska Methodist Hospital, Omaha 3. Meetings third Wednesday of each month at 6 P.M. in either Omaha or Lincoln.

#### New England

**NEW ENGLAND ROENTGEN RAY SOCIETY.** *Secretary-Treasurer,* George Levene, M.D., Massachusetts Memorial Hospitals, Boston. Meets monthly on third Friday at Boston Medical Library.

#### New Hampshire

**NEW HAMPSHIRE ROENTGEN SOCIETY.** *Secretary-Treasurer,* Albert C. Johnston, M.D., Elliot Community Hospital, Keene. Meetings quarterly in Concord.

#### New Jersey

**RADIOLOGICAL SOCIETY OF NEW JERSEY.** *Secretary,* Raphael Pomeranz, M.D., 31 Lincoln Park, Newark 2. Meetings at Atlantic City at time of State Medical Society and midwinter in Newark as called.

#### New York

**ASSOCIATED RADIOLOGISTS OF NEW YORK, INC.** *Secretary,* William J. Francis, M.D., East Rockaway, L. I.

**BROOKLYN ROENTGEN RAY SOCIETY.** *Secretary-Treasurer,* Abraham H. Levy, M.D., 1354 Carroll St., Bklyn. 13. Meets fourth Tuesday of every month, October to April.

**BUFFALO RADIOLOGICAL SOCIETY.** *Secretary-Treasurer,* Mario C. Gian, M.D., 610 Niagara St., Buffalo 1. Meetings second Monday evening each month, October to May, inclusive.

**CENTRAL NEW YORK ROENTGEN SOCIETY.** *Secretary-Treasurer,* Dwight V. Needham, M.D., 608 E. Genesee St., Syracuse 10. Meetings in January, May, and October.

**LONG ISLAND RADIOLOGICAL SOCIETY.** *Secretary,* Marcus Wiener, M.D., 1430 48th St., Brooklyn 19. Meetings fourth Thursday evening, October to May, at 8:45 P.M., in Kings County Medical Bldg.

**NEW YORK ROENTGEN SOCIETY.** *Secretary,* Wm. Snow, M.D., 941 Park Ave., New York 28.

**QUEENS ROENTGEN RAY SOCIETY.** *Secretary,* Jacob E. Goldstein, M.D., 88-29 163rd St., Jamaica 3. Meets fourth Monday of each month.

**ROCHESTER ROENTGEN-RAY SOCIETY.** *Secretary,* Murray P. George, M.D., 260 Crittenden Blvd., Rochester 7. Meets at Strong Memorial Hospital, third Monday, September through May.

#### North Carolina

**RADIOLOGICAL SOCIETY OF NORTH CAROLINA.** *Secretary-Treasurer,* James E. Hemphill, M.D., Professional Bldg., Charlotte 2. Meets in May and October.

#### North Dakota

**NORTH DAKOTA RADIOLOGICAL SOCIETY.** *Secretary,* Charles Heilman, M.D., 1338 Second St., N. Fargo.

#### Ohio

**OHIO STATE RADIOLOGICAL SOCIETY.** *Secretary-Treasurer,* Carroll Dundon, M.D., 2065 Adelbert Road, Cleveland 6. Next meeting at annual meeting of the State Medical Association.

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**CINCINNATI RADIOLOGICAL SOCIETY.** *Secretary,* Eugene L. Saenger, M.D., 735 Doctors Bldg., Cincinnati 2. Meets last Monday, September to May.

**CLEVELAND RADIOLOGICAL SOCIETY.** *Secretary-Treasurer,* George L. Sackett, M.D., 10515 Carnegie Ave., Cleveland 6. Meetings at 6:30 P.M. on fourth Monday, October to April, inclusive.

#### Oklahoma

**OKLAHOMA STATE RADIOLOGICAL SOCIETY.** *Secretary-Treasurer,* Peter M. Russo, M.D., 230 Osler Building, Oklahoma City. Meetings three times a year.

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**Oregon**

OREGON RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Wm. Y. Burton, M.D., 242 Medical Arts Bldg., Portland 5. Meets monthly, on the second Wednesday, at 8:00 P.M., in the library of the University of Oregon Medical School.

**Pacific Northwest**

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**Pennsylvania**

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PHILADELPHIA ROENTGEN RAY SOCIETY. *Secretary*, Arthur Finkelstein, M.D., Graduate Hospital, Philadelphia. Meets first Thursday of each month at 8:00 P.M., from October to May in Thomson Hall, College of Physicians, 21 S. 22d St.

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**Rocky Mountain States**

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Maurice D. Frazer, M.D., Lincoln Clinic, Lincoln, Nebr.

**South Carolina**

SOUTH CAROLINA X-RAY SOCIETY. *Secretary-Treasurer*, Robert B. Taft, M.D., 103 Rutledge Ave., Charleston 16.

**Tennessee**

MEMPHIS ROENTGEN CLUB. Meetings second Tuesday of each month at University Center.

TENNESSEE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, J. Marsh Frère, M.D., 707 Walnut St., Chattanooga. Meets annually with State Medical Society in April.

**Texas**

DALLAS-FORT WORTH ROENTGEN STUDY CLUB. *Secretary*, X. R. Hyde, M.D., Medical Arts Bldg., Fort Worth 2. Meetings on third Monday of each month in Dallas in the odd months and in Fort Worth in the even months.

HOUSTON X-RAY CLUB. *Secretary*, Curtis H. Burge, M.D., 3020 San Jacinto, Houston 4. Meetings fourth Monday of each month.

TEXAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, R. P. O'Bannon, M.D., 650 Fifth Ave., Fort Worth 4. Next meeting Jan. 7-8, 1949.

**Utah**

UTAH STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, M. Lowry Allen, M.D., Judge Bldg., Salt Lake City 1. Meets third Wednesday, January, March, May, September, November.

UNIVERSITY OF UTAH RADIOLOGICAL CONFERENCE. *Secretary*, Henry H. Lerner, M.D. Meets first and third Thursdays, September to June, inclusive, at Salt Lake County General Hospital.

**Virginia**

VIRGINIA RADIOLOGICAL SOCIETY. *Secretary*, P. B. Parsons, M.D., Norfolk General Hospital, Norfolk 7.

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WASHINGTON STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Homer V. Hartzell, M.D., 310 Stimson Bldg., Seattle 1. Meetings fourth Monday October through May, at College Club, Seattle.

**Wisconsin**

MILWAUKEE ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, A. Melamed, M.D., 425 E. Wisconsin Ave., Milwaukee 2. Meets monthly on second Monday at the University Club.

RADIOLOGICAL SECTION OF THE WISCONSIN STATE MEDICAL SOCIETY. *Secretary*, S. R. Beatty, M.D., 185 Hazel St., Oshkosh. Two-day meeting in May and one day with State Medical Society, September.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE. Meets first and third Thursdays 4 P.M., September to May, Service Memorial Institute, Madison 6.

**Puerto Rico**

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA.—*Secretary*, Jesus Rivera Otero, M.D., Box 3524, San-turce, Puerto Rico.

**CANADA**

CANADIAN ASSOCIATION OF RADIOLOGISTS. *Honorary Secretary-Treasurer*, E. M. Crawford, M.D., 2100 Marlowe Ave., Montreal 28, Quebec. Meetings in January and June.

LA SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTROLOGIE ET DE RADIOLOGIE MÉDICALES. *General Secretary*, Origène Dufresne, M.D., Institut du Radium, Montreal. Meets third Saturday each month.

**CUBA**

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA. Offices in Hospital Mercedes, Havana. Meets monthly.

**MEXICO**

SOCIEDAD MEXICANA DE RADIOLOGÍA Y FISIOTERAPIA. *General Secretary*, Dr. Dionisio Pérez Cosío, Marsella 11, México, D. F. Meetings first Monday of each month.

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## ROENTGEN DIAGNOSIS

### THE HEAD AND NECK

**Significance of Indentures in the Outlines of the Atria of the Lateral Ventricles After Air Filling.** M. H. Poppel and J. F. Roach. *Am. J. Roentgenol.* 58: 46-50, July 1947.

After air filling of the lateral ventricles an indenture or defect known as the glomus indenture may be seen on the lower wall, just rostral to the subdivision of the lateral ventricular body into an occipital and a temporal horn (atrial region). It is produced by the upward and backward projection of the choroid plexus. Dyke and Davidoff (*Bull. Neurol. Inst., New York*, 2: 331, 1932) considered the normal limits of this defect to be 15 by 15 mm. Enlargement may occur due to trauma incident to puncture of the ventricles for introduction of air, but encephalography a week after such a finding will usually show a return to normal. An enlarged defect on the side contralateral to the ventricular puncture has been shown to be due to retention cysts of the glomus of the choroid plexus. Enlargement may also be due to benign hypertrophy or to other tumors.

A second indenture, known as the calcar avis defect, is produced by an invagination of the ventricular wall in the region of the junction between the atrium and the occipital horn on the medial and slightly on the dorsal aspect of this portion of the ventricle. It varies greatly in size and is not necessarily the same size on both sides. Care must be exercised not to regard this indenture as pathological but to classify it as a variation in the normal. CLARENCE E. WEAVER, M.D.

**Parathyroid Adenoma: A Diagnostic Case Study.** Harold H. Joffe, F. H. Magney, and Arthur H. Wells. *Minnesota Med.* 30: 760-764, July 1947.

A patient with a past history of an alcohol injection for trigeminal neuralgia and the curettement of a giant-cell tumor of the os calcis was admitted for fracture of the femur which occurred through a cystic lesion also suggestive of giant-cell tumor. She was later admitted for pain in the right knee. Roentgen examination at that time revealed cystic areas in the patella and other bones and a renal stone on the right. The serum calcium was elevated; the phosphorus normal. An adenoma was removed from the left superior parathyroid gland.

The authors review the literature and discuss the pathologic physiology of the parathyroid gland and the symptoms and differential diagnosis of hyperparathyroidism. The relatively slight elevation of blood calcium is stressed, and the fact is pointed out that cases of renal lithiasis may exhibit hypercalcinuria without hyperparathyroidism. Stress is also laid on the point that any case of supposed giant-cell tumor should be carefully investigated to rule out a possible hyperparathyroidism. PERCY J. DELANO, M.D.

**Deafness, Tinnitus, Vertigo, and Neuralgia.** David J. Goodfriend. *Arch. Otolaryng.* 46: 1-35, July 1947.

Abnormalities of the dental bite have been shown to cause abnormalities of the structure and movements of the mandibular joint and to alter its relationships to the external auditory meatus, the middle ear, and the eustachian tube so as to cause deformity, injury, and

degeneration of their walls and to disturb their blood and nerve supplies, with resultant auditory disturbances and neuralgias. The present study represents the co-operation of the dentist, the otolaryngologist, the anatomist, the radiologist, and the psychologist. Only the radiologic aspects of the study will be covered in this abstract.

Pendergrass and Kornblum devised a roentgenologic technic for the study of the mandibular joint, and on this basis the author standardized normality and typed some abnormalities. Roentgenograms of the mandibular joints are accurate aids in the diagnosis of joint and bite abnormalities when correlated with the mounted reproductions of the teeth, the facial measurements, and palpation of the joints. The roentgenologic technic is not complex and requires no lamination or other special procedures. With the cassette held close to the joint of which a roentgenogram is to be taken, the ray is directed from the opposite side at an angle of 20 degrees from the horizontal axis of the upright head and cassette. Views are taken of each joint, one with the bite closed and one with a 1-inch (2.5-cm.) block held between the front teeth. For further simplification of the technic, the author has devised a ray director consisting of a plastic cassette holder from the top of which a clear rod projects at an angle of 70 degrees. This rod is therefore 20 degrees from the horizontal of the upright head and cassette. The roentgen rays are so directed that they are parallel to the projecting rod.

Roentgenograms of normal and abnormal mandibular joints are reproduced.

**Applications of Methods of Salivary Gland Examination to the Diagnosis of Neoplastic Affections of the Parotid and Submaxillary Regions.** G. F. Leroux. *J. de radiol. et d'électrol.* 28: 85-94, 1947.

This article begins with a historical note and a discussion of the various solutions which have been employed for demonstration of the salivary glands. The author favors a solution of the lipiodol type. The trend of the discussion is along quite systematic lines, and a large number of criteria for diagnosis of neoplastic alterations in a normal pattern are set down. The article might be more convincing if the illustrations were more lucid.

The greatest flaw in all such presentations lies in the fact that to make assertions of diagnostic value about any film, it is most necessary that one have a background developed by the scrutiny of large numbers of normal exhibits. Very few men have such a background when dealing with roentgen "sialography" and consequently are in not much of a position to make critical observations. PERCY J. DELANO, M.D.

### THE CHEST

**"Dispersed" Bronchiectasis.** P. Santy, Marcel Bérard, Galy and Pierre Fraisse. *J. franç. de méd. et chir. thoracique* 1: 4-9, February 1947.

This paper is based on a series of 50 cases of bronchiectasis. Cylindrical bronchiectasis of small caliber confined to a single lobe should be treated medically;

all the others are surgical cases. For the roentgen demonstration of the lesions at least two views should be taken. Before the injection of lipiodol, medication with sulfa drugs or penicillin and postural drainage are recommended to clear the bronchi of mucus.

The authors distinguish three types of bronchiectasis; strictly lobar, diffuse, and scattered or "dispersed." The majority of their cases were of the diffuse type. They are particularly interested, however, in the third type. This resembles the "single dilatation" type described by Chapman and Wiggins (*Ann. Int. Med.* 14: 2047, 1941). It involves bronchi of the third order, and because of the variability of the location the authors suggest for it the designation "dispersed." The etiology is not known but it is believed that a localized mediastinitis is the most probable explanation.

Excellent illustrations enhance the value of this article.

EUGENE F. LUTTERBECK, M.D.

**Additional Case of Bronchiectasis Formation Below a Cavity.** P. Pruvost, Depierre and Kerbrat. *J. franç. de méd. et chir. thoracique* 1: 156-160, April 1947.

The so-called "Ameuille" syndrome, first described by Ameuille and Mezard in 1933, is found in patients with chronic tuberculosis. Its onset is characterized by sudden, severe pain in one side, and the roentgenogram reveals a large area of increased and homogeneous density suggesting an atelectasis with obstruction. According to Ameuille, the course of events is as follows: first cavity formation, then retractile consolidation of the lung, and finally the appearance of bronchiectasis.

In the cited case, of a 15-year-old boy, the above symptoms were all observed, but in a different order. The diagnosis of bronchiectasis was made long before the sudden advent of the retractile consolidation of the lung, and the gradual development of a cavity came about afterward.

The authors discuss the development of bronchiectasis before cavity formation. Their patient had clubbed fingers and repeated coughs in early childhood. After the incident of retractile consolidation, all the clinical symptoms became much worse. It is emphasized that in spite of the extensive and homogeneous density of the lung, no obstruction of the bronchus could be found with the bronchoscope.

The prognosis in these cases is hopeless and treatment is solely symptomatic.

The article is illustrated, showing by use of bronchography and tomography the various stages of the pulmonary lesion.

EUGENE F. LUTTERBECK, M.D.

**Segmentary Bronchitis of the Middle Lobe Masking a Bronchial Cancer.** E. Rist and J. M. Lemoine. *J. franç. de méd. et chir. thoracique* 1: 161-165, April 1947.

The authors in collaboration with Ameuille have previously reported 7 cases of so-called "segmentary bronchitis." At this time they report 2 cases, where a localized infiltration of the right middle lobe was accompanied not only by an inflammatory process but also by a slow growing carcinoma.

The first patient was a 63-year-old man whose symptoms were of some seven years' duration. A triangular area of irregular density was found in the right middle lobe. The patient died at a second admission to the hospital; surgery was not attempted and no autopsy was done.

The second case is that of a 54-year-old man who had attacks of slight cough and elevated temperatures for two years. A rather homogeneous density was found in the right middle lobe. The patient died postoperatively and autopsy confirmed the diagnosis of malignant tumor of the right middle lobe.

The authors emphasize the importance of watching for early evidences of cancer in cases of segmentary bronchitis, where the inflammatory process of the lungs might be superimposed.

EUGENE F. LUTTERBECK, M.D.

**Bronchography.** J. M. Dell, Jr. *South. M. J.* 40: 543-549, July 1947.

The author's technic of bronchography is as follows: Following preliminary medication with nembutal, codeine, and atropine, the pharynx is anesthetized with 2 per cent pontocaine. The vocal cords are sprayed with pontocaine, with the aid of a syringe with a laryngeal cannula, and 2 c.c. of pontocaine are permitted to trickle down into the trachea. A 14-F urethral catheter is then passed through the nasal passages down into the trachea to a point 2 inches above the carina. The position of the catheter is checked fluoroscopically and, with the patient in varying positions, iodized oil is then injected through it. Both lower lobes, the right middle lobe, the lingular portion of the left lower lobe, and part of the left upper are visualized. In order to visualize the upper lobe branches, a separate study is done with the catheter in the main bronchus.

Complications to bronchography are minimal and consist of iodism, reactions to the anesthetic agent, and attacks of asthma. In extremely rare instances death has been reported.

MORRIS IVKER, M.D.

**Pulmonary Tuberculosis in Chinese Students.** J. C. Tao. *Am. Rev. Tuberc.* 56: 22-26, July 1947.

Fluoroscopic examination of the chests of 17,155 high school and university students in Chungking was done during the period from May 1944 to March 1946. All those with suspicious shadows were re-examined after two weeks and roentgenograms were made. At this time a clinical history was obtained, physical examination was carried out, and erythrocyte sedimentation rate and sputum studies were completed. Among those examined, 1,184 (6.9 per cent) were found to have active pulmonary tuberculosis, which was asymptomatic in more than 80 per cent; 71 per cent of the lesions were classified as minimal and 27 per cent as moderately advanced.

L. W. PAUL, M.D.

**Inhibition of Primary Tuberculosis by BCG. A Study in Children, Based on 13,470 Chest Roentgenograms.** Irwin S. Neiman and Erhard Loewinson. *Am. Rev. Tuberc.* 56: 27-35, July 1947.

This study reports the results of BCG vaccination in a group of 1,417 children with another group of 1,414 children serving as controls. Vaccination was done four to six days after birth. All children were observed at fairly regular semiannual intervals from birth, at which times they were subjected to a physical examination, tuberculin test, and chest roentgenography. Up to the present time 13,470 chest roentgenograms have been obtained. The results reveal a significantly higher incidence of primary tuberculosis in the non-

vaccinated children. As an example, in the vaccinated group there have been observed 14 cases with initial lesions, 8 of which subsequently cleared, 4 calcified, and 2 underwent questionable calcification. In the non-vaccinated group, there were 42 initial lesions, 10 of which later disappeared, 18 calcified, (4 questionably), 12 were still active, and 2 of the children died.

L. W. PAUL, M.D.

**Pulmonary Emphysema and Tuberculosis: A Roentgenological and Pathological Study.** Albert Gugenheim. *Am. J. Roentgenol.* 58: 64-74, July 1947.

Emphysema associated with pulmonary tuberculosis is as manifold pathologically as in other chronic pulmonary diseases which are characterized by fibrosis, destruction of lung tissue, and especially by endobronchial changes. In the majority of cases the emphysematous changes are not diffuse but are localized in the area of the tuberculous lesions. In *intrafocal emphysema* bronchial and bronchiolar obstruction play the most important part. *Perifocal emphysema* is by far the most common type. Emphysematous change is almost always present in the immediate vicinity of fibrotic and productive lesions, and to a much lesser degree around exudative lesions. In the periphery of calcific foci it is hardly ever absent. With *emphysematous bullae and blebs*, it is felt that small narrowed bronchi still penetrate their walls allowing air to enter, but preventing it from leaving during expiration. *Compensatory emphysema* may occur in relatively normal portions of the lung when other portions are involved by tuberculosis and loss of respiratory surface ensues.

In primary tuberculosis the calcified Ghon focus may be suspended in emphysematous lung tissue as in a spider web. Obstructive emphysema may result from a caseating lymph node eroding through the wall of a bronchus, creating a valve-like mechanism.

After collapse therapy, hypertrophic emphysema occurs in many cases. This is not always compensatory. There are frequently displacement and torsion of mediastinal structures, and a partial fixation of the bony thorax. The resulting changes in the bronchial tree contribute to the formation of hypertrophic emphysema. In the aged, atrophic emphysema and tuberculosis may coincide.

It is felt that pure compensation cannot be the sole cause of hypertrophic emphysema. There are two conditions which are almost always found in association with it: (1) bronchial and bronchiolar obstruction, and (2) vascular changes in the lung. The vascular changes are the result and not the cause of the emphysema. Capillaries are stretched, their lumina are narrowed and often obliterated. In atrophic emphysema atheromatous and sclerotic changes in pulmonary vessels are of primary importance.

Emphysema occurs during all stages of pulmonary tuberculosis. It is of characteristic appearance in many cases of hematogenous tuberculosis.

CLARENCE E. WEAVER, M.D.

**Pulmonary Pseudo-Cysts Associated with Pneumonia.** L. J. Flax, M. M. Ginsburg, and C. J. Stettinheimer. *Rocky Mountain M. J.* 44: 532-534, July 1947.

True congenital lung cysts are extremely rare. During fifty years not one instance was found in the autop-

sies at the Babies' Hospital in New York City. It is now apparent that many of these so-called cysts are examples of emphysematous cavities. Such cavities may also develop in the course of a pneumonia, when a portion of the bronchial tree becomes occluded by inflammatory exudate and mucosal swelling and the formation of a check-valve obstruction. In infected areas fluid may be found. In 2 cases followed by the authors, large cavities whose appearance suggested congenital cysts disappeared under observation.

A classification of inflammatory pseudo-cysts is included.

PERCY J. DELANO, M.D.

**Massive Atelectasis Following Tonsillectomy Under Local Anesthesia. Report of a Case.** Leroy L. Sawyer. *Arch. Otolaryng.* 46: 45-51, July 1947.

After a brief review of the literature on the pathology and physiology of postoperative collapse of the lung, the author reports a case of massive atelectasis following tonsillectomy under local anesthesia in a 25-year-old woman. A roentgenogram taken two weeks previous to the operation showed the hilar shadows on the right side to be slightly enlarged. The morning following tonsillectomy the patient complained of severe pain in the right side of the chest and some coughing; she had a temperature of 101.2° F., a respiratory rate of 32, and a pulse rate of 108. Physical examination at this time revealed diminution of breath sounds and of the whispered voice over the entire right lung, with decreased tactile fremitus and an impaired percussion note. Roentgenography showed collapse of the right lung, the margin of the heart well over to the right, and the intercostal spaces narrowed, with elevation of the right side of the diaphragm. On bronchoscopy a few hours later, a bloody mucous plug was found in the right main bronchus; this was aspirated. At the end of twenty-four hours, x-ray examination revealed that the lung was about 50 per cent aerated, and at seventy-two hours re-inflation was practically complete. Roentgenograms taken five months and a year after tonsillectomy were normal.

**Report of an Outbreak of Q Fever at the National Institute of Health. I. Clinical Features.** Charles G. Spicknall, Robert J. Huebner, James A. Finger, and William P. Blocker. *Ann. Int. Med.* 27: 28-40, July 1947.

This paper is a clinical report of 45 of 47 cases of Q fever occurring in the first half of 1946 in the National Institute of Health. In only 13 of the cases was there clinical or roentgen-ray evidence of pneumonitis. Chest films in the pneumonitis cases were similar to those described elsewhere. In a few cases the consolidation observed was like that of lobar pneumonia; the changes in other cases could not be differentiated roentgenologically from those caused by atypical pneumonia.

**Pneumoconiosis in Coal Workers in Wales.** J. Gough. *Occup. Med.* 4: 86-97, July 1947.

As in other parts of the world, the incidence of pneumoconiosis in Wales is highest in anthracite workers, but it is also found with disturbing frequency in semibituminous coal miners. The disease is not confined to men working underground but occurs also in those screening coal on the surface of the mine and even in those loading coal into ships, who have never worked

in mines. A small percentage of the affected miners show silicosis of the classic type but these are men who have engaged in drilling rock. In the large majority the changes differ in detail from silicosis, although the distribution in the lung is similar. Two main forms are seen, the one consisting of massive fibrosis and the other of a scattered focal condition which is a simple pneumoconiosis.

Massive fibrosis and classic nodular silicosis in the coal miner present comparatively little difficulty in roentgen diagnosis, but in recent years a new category of disease has been introduced in British law. Since 1943 a roentgen appearance called reticulation has been recognized as indicating a form of pneumoconiosis for which compensation may be awarded, and on account of it a large number of men have been certified and excluded from the mines. Roentgen-ray reticulation consists of small opacities which may appear as separate granules or may seem to be linked up into an ill-defined network. There are many variations in the pattern, and the opacities are often blurred and irregular in outline, lacking the density and sharpness seen with silicotic nodules.

When one compares the pulmonary changes seen in sections of the lung with the roentgen appearances, it is found that the focal lesions give reticulation whether emphysema is present or not, and it is difficult to determine by roentgen rays how much emphysema is present in a particular case. Since emphysema is probably the most disabling component, it follows that roentgenology does not provide reliable criteria for the assessment of disability. This difficulty in estimating the severity of disease is one of the most urgent problems needing solution.

**Contributions by Dr. Gardner to the Pathology of Bauxite Workers' Lung.** Andrew Rutherford Riddell. *Occup. Med.* 4: 56-67, July 1947.

One of the last of the pneumoconioses to engage the attention of the late Dr. Leroy U. Gardner was that occurring in bauxite workers. He studied the material obtained at necropsy in 2 cases reported here. The first patient spent practically all his working lifetime in dusty trades, and for the last eleven years had been employed around furnaces in a corundum manufacturing plant. He finally had to stop work because of "heart disease." He was found in poor physical condition, with clinical and roentgen evidence of pneumothorax. No evidence was found to indicate that this man was suffering from tuberculosis or any other disease of infective origin. He never experienced severe thoracic pain, but complained of a "tightness" and "pulling in" sensation in his chest and of the occurrence of some "bad spells," when he had great difficulty in getting his breath. He died about six months after he ceased work, at the age of fifty-five.

The pathologic picture was unlike anything previously encountered. It was characterized by broad bands of pigmented scar tissue located chiefly in the depths of the lung, with a secondary emphysema. The cause of this condition is not apparent. The lesions of beryllium workers—small, discrete, cellular granulomas which tend to heal with hyaline fibrosis—were altogether lacking in this case. The pattern was not that of any of the well known varieties of pneumoconiosis which are associated with fibrosis. The absence of nodules, characteristic of silicosis, would appear to ex-

clude that condition; the absence of the characteristic bodies would eliminate the possibility of asbestosis.

The most unusual feature about the mineralogic data in this case was the failure of roentgen rays to account for about nine tenths of the total silica in the ash of the lung, which would mean either that this silica existed in amorphous state or that it was largely composed of materials like clay minerals which may be broken down by acid treatment. The latter possibility was eliminated by further tests. The other alternative, that the remainder of the silica existed in amorphous state which gives no characteristic lines in the roentgen ray diffraction pattern, has not been definitely excluded.

The second case was that of a man who had also been in contact with arc furnaces. Clinical examination and a roentgenogram of the chest showed a condition similar to that observed in the first case. The patient died at the age of forty-two, about a year and a half after he was forced to stop work because of illness. The chemical and roentgen ray diffraction analyses, like the histologic picture, were almost identical with the findings in the first case.

**Nodulation with Superimposed Infection in Lungs of Foundry Grinders and Burners.** Report of Four Unusual Cases. L. E. Hamlin. *Occup. Med.* 4: 111-121, July 1947.

Findings in the roentgenograms of the chests of 4 employees in the foundry industry showing suggestive evidence of tuberculosis are presented to demonstrate the unusual course of the infection in the presence of nodulation hitherto presumed to be due to silicosis. The progress of the disease is inconsistent with the usual picture of tuberculosis superimposed on silicosis, both from the clinical and the roentgenographic aspect. Occupational exposure as demonstrated by industrial hygiene surveys in the plants where these roentgen patterns were produced did not appear to be adequate to produce the extensive nodulations observed but did seem to be sufficient to cause siderosis. The unorthodox behavior of both infection and nodulation, in spite of continuous exposure, substantiates the impression that the roentgenographic appearance is the result of the deposition of iron pigment (siderosis) rather than reaction in pulmonary tissue due to contact with free silica.

**Hilar Cysts.** P. Santy, M. Bérard, and P. Galy. *J. franç. de méd. et chir. thoracique* 1: 66-90, March 1947.

After reviewing the literature and discussing the various types of mediastinal cysts according to their histologic structure and typical position, the authors report 4 cases of hilar cyst (*kyste du pédicule pulmonaire*).

Hilar cysts are of bronchogenic origin and are found for the most part in the anterior mediastinum. They are of variable size. Many of them have direct bronchial or blood vessel connection with the hilar structures. The liquid content varies with the degree of infection. The clinical manifestations are also dependent upon the amount of infection and upon the growth of the cyst. Hemoptysis occurred in 2 of the authors' cases.

For the diagnosis of these cysts it is strongly recommended that roentgenograms be obtained in various projections and that use be made of tomography and of the fluoroscope to detect changes in the shape of the cyst during respiration. In one case examination in the Trendelenburg position demonstrated the mobility of

the cyst. Pneumothorax has also been of great diagnostic value, as has pleuroscopy. Differentiation must be made from bronchial carcinoma, hydatid cyst, and non-parasitic cysts of the lung.

Because of the danger of enlargement and infection, operation is indicated. The results in the cases reported were excellent. EUGENE F. LUTTERBECK, M.D.

**Coarctation of the Aorta.** Maurice Campbell and S. Suzman. *Brit. Heart J.* 9: 185-212, July 1947.

The authors report a study of coarctation of the aorta and present a new sign for its recognition, namely an increase in the visible collateral circulation when the patient bends forward with the arms hanging vertically. Of other signs, notching or scalloping of the ribs is almost pathognomonic of coarctation, and films are reproduced showing that such notching may appear as early as seven years of age, becoming increasingly prominent in successive examinations. With the constantly increasing number of roentgen surveys of supposedly normal persons, this sign should be more commonly encountered. The aortic knob may or may not be demonstrable on the roentgenogram, but the descending aorta will not be seen. There may or may not be demonstrable enlargement of the left ventricle.

Among the cardinal points in the clinical diagnosis is a raised blood pressure in the upper half of the body with low blood pressure in the legs and feeble or absent pulsation in the femoral arteries and the abdominal aorta. The diagnosis is most likely to be made in the less obvious cases if the femoral pulse is felt for in every patient with high blood pressure or with obvious arterial pulsation in the neck, or with an unexplained systolic (or diastolic) murmur at the base of the heart.

Since coarctation of the aorta is one of the congenital anomalies amenable to surgery, we should be on the alert to diagnose it. Without operation life expectancy is considerably below normal.

ZAC A. ENDRESS, M.D.

**Symmetrical Double Aortic Arch. Report of a Case.** Dean K. Crystal, Henry W. Edmonds, and Paul F. Betzold. *West. J. Surg.* 55: 389-392, July 1947.

Vascular rings which encircle the trachea and esophagus may produce difficulty in respiration and deglutition. There is a fairly characteristic syndrome with stridor, wheezing, crowing, bouts of cyanosis, dysphagia, regurgitation, and recurrent respiratory infections. The feasibility of interrupting surgically some portion of the inelastic vascular ring has focused attention on these anomalies.

The aorta normally develops from the fourth left branchial artery of the early embryo. If the fourth right arch persists as the continuation of the ascending aorta, the aortic arch will lie wholly to the right of its normal position and to the right of the trachea and esophagus. This will not be of consequence if there is accompanying situs inversus of thoracic viscera, but when the arch is on the right and the descending aorta is in its normal position on the left, constriction may occur. The junction of the arch and descending aorta will then lie posterior to the esophagus, in which it will produce a constant indentation readily demonstrable roentgenologically.

In cases of right aortic arch, constriction may occur in three ways: first, the ductus arteriosus (or ligamentum arteriosum) may comprise the right lateral seg-

ment of a circle as it extends from the aorta to the left pulmonary artery. Second, a rudimentary left aortic arch may be present in front of and to the left of the trachea in combination with a larger persistent right arch to which the left unites posteriorly. Third, double symmetrical right and left aortic arches persist, which embrace the trachea and esophagus tightly and unite posteriorly to form the descending aorta.

The case reported by the authors was of the third type. When first seen, the patient was eight weeks old, with a history of wheezing and dyspnea since birth. Each breath was accompanied by a loud crow or wheeze and there was marked respiratory effort including use of accessory muscles. Attacks of mild dyspnea and cyanosis changed rather rapidly to apnea and unconsciousness lasting from thirty seconds to ten minutes.

Early studies—bronchoscopy and barium through a catheter which extended nearly to the cardia—showed no abnormality. At the age of ten months the child began to have convulsions following the apneic attacks and showed distention of the superficial veins of the torso and neck. A roentgen study at this time, with barium by mouth, demonstrated a constant indentation of the esophagus from behind at the level of the 2d and 3d dorsal vertebrae. This was interpreted as due to the aortic arch passing behind the esophagus. The symptoms could be explained on the assumption of (a) a persistent right aortic arch, (b) the descending aorta on the left, and (c) constriction of the esophagus and trachea by a left aortic arch or the ligamentum arteriosum or both.

At operation the pericardium was found to extend unusually high above the heart and it was necessary to incise it to visualize the atretic ductus arteriosus, which was ligated and cut. The left subclavian was visible lateral to the trachea. The left carotid was palpable behind the sternum. The descending aorta could be felt behind the esophagus. It was considered unwise to incise the pericardium further because of its intimate attachment to the trachea, and the chest was closed. The patient died two hours later.

At autopsy, the reason for failure of surgery was shown. There were symmetrically placed right and left aortic arches which tightly embraced the trachea and esophagus. From each arch the carotid and subclavian arteries arose for the respective side. The arches united posteriorly to form the descending aorta. The pericardium was attached above to the arches, and the left arch, which was missed at operation, lay in the fold of pericardium which seemed so adherent to the trachea.

B. S. KALAYJIAN, M.D.

**Four Important Congenital Cardiac Conditions Causing Cyanosis to Be Differentiated from the Tetralogy of Fallot: Tricuspid Atresia, Eisenmenger's Complex, Transposition of the Great Vessels, and a Single Ventricle.** Fred Alexander and Paul D. White. *Ann. Int. Med.* 27: 64-83, July 1947.

Four cases are presented to illustrate four congenital cardiac conditions causing cyanosis which require differentiation from the tetralogy of Fallot.

**Tricuspid Atresia:** Clinically certain features in combination afford a clue to the diagnosis of tricuspid atresia: cyanosis, usually noted at birth; a left axis deviation on the electrocardiogram; a left-sided enlargement of the heart demonstrable roentgenographically; polycythemia (with clubbing in some instances); a loud

systolic murmur, best heard in the second and third interspaces just to the left of the sternal line. The authors' patient was a boy of five and one-half months in whom all these features were present. Other congenital anomalies of a compensatory nature, as patent ductus arteriosus, interventricular septal defect, patent foramen ovale, and transposition of the great vessels are frequently associated with tricuspid atresia.

**Eisenmenger's Complex:** The Eisenmenger complex consists of dextroposition of the aorta, interventricular septal defect, and right ventricular hypertrophy, with a pulmonary artery which may be normal or dilated. It is the condition of the pulmonary artery which distinguishes this complex from the tetralogy of Fallot, the latter being invariably accompanied by a certain degree of pulmonary stenosis. Clinically the following factors appear rather constant in this complex: a definite, distinct prominence of the pulmonary conus with a "dance" of the hilar shadows demonstrable by fluoroscopy and right axis deviation on the electrocardiogram. The systolic murmur may be variable. There are less cyanosis and clubbing than in the tetralogy of Fallot. At times, hoarseness is noted due to pressure of the pulmonary conus on the recurrent laryngeal nerve. Dyspnea may be present, as well as the other general symptoms of congenital heart disease, such as cough, polycythemia, dysphagia, abnormal susceptibility to infections, weakness, dizziness, convulsions, and tingling of the extremities.

The authors' patient was a boy of five years. The diagnosis was based on the physical findings, the roentgen film, fluoroscopic evidence of pulmonary congestion, and right axis deviation in the electrocardiogram. Surgery was avoided for fear of further burdening an already overloaded pulmonary circulation.

**Transposition of the great vessels** may at least be suspected when a goodly number of the following findings are present: growth difficulties, cyanosis and dyspnea, spells of fainting, increased red blood count, a variable murmur (apical systolic at times), enlargement of both ventricles demonstrable on x-ray examination right axis deviation on the electrocardiogram, and such minor signs as enlargement of the liver and spleen, edema of the extremities, or choking cough. Surgery is obviously of no benefit in this type of case and should be avoided after the correct diagnosis is made. The authors' patient died seven and a half hours after an attempted operation for tetralogy of Fallot.

**Single Ventricle:** Complete cor biloculare is a rare finding, either with division of the truncus arteriosus as in the case reported, or without division. Clinical recognition of this congenital abnormality as a single entity would appear to be impossible, although a few findings bear mention in this respect. First, some degree of cyanosis is usually found. Generally a systolic murmur is heard over the entire precordium. Usually a rather definite degree of right axis deviation is shown in the electrocardiogram and an enlarged globular heart in the roentgen film. Obviously surgery is more detrimental than helpful in this type of anomaly.

STEPHEN N. TAGER, M.D.

**The Blalock-Taussig Operation.** George W. Salmon and Howard T. Barkley. *J. Pediat.* 31: 31-54, July 1947.

The authors report their experiences with the Blalock-Taussig operation. The chest was explored in 10 pa-

tients, and anastomosis was done in 8, 6 of whom were markedly improved clinically. The other 2 died within twenty-four hours.

The most constant physical findings, according to the authors' report, were delayed cyanosis, syncopal attacks followed by an increase in cyanosis, and a loud systolic murmur and thrill over the left sternal border, best heard in the second and third interspaces. The heart size was usually within normal limits, but there was an absence of hilar pulsations with a decrease in the size of the pulmonary arteries as viewed in the right anterior oblique position. The electrocardiogram showed right axis deviation.

Following anastomosis there occurred a slight increase in the heart size (as determined by the cardiothoracic ratio) and in most cases there was also an increase in the size of the pulmonary conus segment. Electrocardiograms showed no change.

The authors feel that a 20 per cent mortality, as shown by their series, is not prohibitive when the severity of the disease is considered and when it is remembered that in 80 per cent an appreciable number of years have been added to the life expectancy of these children.

J. FURNARI, M.D.

## THE DIGESTIVE SYSTEM

**Hiatus Herniæ (A New Method of Demonstrating Hiatus Herniæ Radiologically).** Joseph Bloom. *Canad. M. A. J.* 57: 9-13, July 1947.

Bloom discusses hiatus hernia from the standpoint of etiology, incidence, predisposing factors, habitus, clinical symptomatology, and radiological features. Hiatus hernia may be due to congenital weakness or maldevelopment of the diaphragm, to senile atrophic changes of the periesophageal tissues of the diaphragm, to increased intra-abdominal pressure, or to malformation of the thoracic or lumbar spine leading to kyphosis or scoliosis and consequent shortening of the skeletal structure. The condition is believed by some to be more common in females but the ratios differ in different series. It has been said to occur chiefly in persons over fifty years of age, but the author believes the incidence in younger individuals to be greater than is usually reported.

Hiatus hernias may be asymptomatic or the patient may complain of pain coming on after meals and usually relieved on assumption of the erect posture. Dysphagia occurs frequently, particularly with complicating esophagitis or ulceration. Pulmonary symptoms, as cough and dyspnea, are usually present only in association with large herniations. Belching, regurgitation, and vomiting are common. Cardiac symptoms with typical anginal pain occur. Fainting spells are not infrequent. Electrocardiographic findings may suggest myocardial disease, but the cause and mechanism of these changes are still not well understood. Hematemesis, melena, and anemia have been observed. Frequently associated diseases are peptic ulcer, cholelithiasis, herniations elsewhere, diverticulosis of the colon or duodenum, and scoliosis or kyphosis of the thoracic spine.

The routine chest film may or may not reveal the presence of a hiatus hernia. The author uses a procedure which he believes has not previously been described. After the usual examination of the duodenum and stomach, the patient is given a mouthful of barium and then placed in the horizontal or slight Trendelen-

burg position. He is then positioned in the right posterior semi-oblique, which affords a clear view of the posterior mediastinum, and is instructed to swallow the barium. When the esophagus is completely filled, he is told to take a deep breath and hold it. If a herniation of the cardia is present, it will come into view along with any diverticula of the esophagus which may not have been demonstrated earlier. A dilated esophageal ampulla will also be shown by this procedure. Spot films are taken of the hernia site.

The mechanism of this procedure is explained as follows: The deep breath taken after the esophagus is filled with barium "causes contraction of the diaphragms and their crura, thus widening the oesophageal hiatus; it also causes an increase in the intra-abdominal pressure and a decrease in the intrathoracic pressure. The barium-filled oesophagus acts as a drainage tube ready to obey the laws of gravity and fluid seeking its own level; in other words, acting as a syphon drainage tube to empty the stomach contents into and through the oesophagus. This syphon action, aided by the increased intra-abdominal pressure and the diminished intra-thoracic pressure, sucks the cardia portion of the stomach through the lax oesophageal hiatus, allowing the latter to manifest itself as a herniation above the diaphragm."

Six small reproductions illustrate hiatus hernias associated with other pathological conditions for which they may be mistaken. WILLIS MANGES, M.D.

**Oesophageal Hiatus Hernia.** John G. Stapleton. *Canad. M. A. J.* 57: 13-16, July 1947.

Radiology affords the most practical and easiest way to diagnose esophageal hiatus hernias. A high percentage of cases, however, are not diagnosed until the second or third radiologic study. This the author believes is purely a matter of technic.

Esophageal hiatus hernias are classified into two main groups according to the position of the esophagus: In Group I, the para-esophageal group, a portion of the stomach has protruded into the posterior mediastinum beside the esophagus. The hernias in this group are usually small and only rarely is a large portion of the stomach involved. Group II consists of those hernias in which the esophagus is displaced upward into the posterior mediastinum along with the herniated portion of the stomach. The extra length of the esophagus is taken up by its increased tortuosity. This type of hernia may become very large and include colon or omentum. To these two groups may be added a third—namely, the congenital short esophagus type. Here the esophagus is too short to reach the diaphragm and the stomach has always been partially within the thorax.

The cause of esophageal hiatus hernias is related to increased intra-abdominal pressure, usually as a result of obesity or pregnancy. The muscle fibers, including those of the diaphragm, lose their tone with increased age and this may be the reason for the increased incidence of hernias in older patients.

During the radiologic examination the patient should be given a routine drink of barium in the decubitus position, since only a few hernias are visible on erect fluoroscopic examination. Effort is made to demonstrate the hernia on films but routine technic for increasing the abdominal pressure is not used because it is felt that hernias which require increased abdominal pressure to

make them visible are probably of no clinical significance.

The roentgen diagnostic points mentioned are: (1) the presence of a sac-like globular mass of barium above the diaphragm; (2) a slight constriction just above the globular barium mass, which represents the site of junction of the esophagus with the stomach; (3) a narrowed area below the globular barium mass, which represents the area where the thoracic portion of the stomach passes through the diaphragm; (4) gastric mucosal pattern in the thoracic cavity; (5) an asymmetrical relationship of the esophagus with the barium mass.

Large hernias should be repaired surgically if the patient has symptoms and is a good operative risk. Medical treatment is advised for those patients with minor symptoms and for those who are poor operative risks. JOHN DECARLO, M.D.

**Management of Cardiospasm.** Rudolph Schindler. *California Med.* 67: 23-28, July 1947.

The treatment of choice for cardiospasm is a brusque dilatation of the spastic esophagus and rupture of the musculature. This may be done by introduction of a hydrostatic or pneumatic bag, but the author prefers a metal dilator, which is spread in umbrella-like fashion after it is in the cardia. Of 26 cases treated by this method, 24 were cured. There was 1 failure and 1 recurrence.

The first step in the procedure is a careful x-ray study. The picture is usually characteristic. The esophagus is dilated and tortuous; its lower contour is perfectly smooth, with no filling defect. The barium may be stopped within the hiatus of the diaphragm or in the cardia. Since the dilating instrument must be exactly within the constriction, it becomes necessary to measure the exact distance from the teeth to the point of narrowing. This should be done under fluoroscopic control with the instrument itself. An organic lesion must be excluded by gastroscopy.

Brief mention is made of other therapeutic measures: psychotherapy, effective only in early cases; introduction of bougies; and surgery, which may exceptionally be required. MAURICE D. SACHS, M.D.

**Clinical Significance of Diverticulosis, Including Diverticulitis, of the Gastrointestinal Tract.** Emile C. Nash and Walter Lincoln Palmer. *Ann. Int. Med.* 27: 41-63, July 1947.

Diverticula are found in all parts of the digestive tract. *Esophageal diverticula* are of two types, pulsion and traction. The pulsion diverticulum, known also as the pharyngo-esophageal diverticulum, is a true herniation of the mucosa and submucosa through the fibers of the inferior constrictor muscles of the pharynx as they run transversely or through the obliquely dividing fibers of the cricopharyngeus muscles on the posterior aspect of the esophagus. The symptoms are directly referable to the act of swallowing, dysphagia is first noticed with dry foods, increasing gradually in magnitude until finally water and other liquids are regurgitated. These diverticula are readily demonstrable roentgenologically.

Traction diverticula usually develop in the region of the left main bronchus or near the cardia. There appears to be no characteristic symptomatology. In 7 of 20 cases selected at random, the presenting symptoms

were fairly typical of an associated organic disease, as cholelithiasis, gastric or duodenal ulcer, carcinoma of the stomach, and pleural effusion. Symptoms referable to the esophagus in cases without coexistent disease were substernal burning, discomfort after eating, and intermittent dysphagia.

Gastric diverticula are of less common occurrence than those of the esophagus. About 65 per cent occur near the cardia. Here their roentgenologic diagnosis may be difficult, requiring a careful search in various positions.

In the duodenum, the differentiation of congenital diverticulum and the pseudodiverticulum produced by a chronic ulcer is a problem. Congenital diverticula are rare in the duodenal bulb, whereas almost all of the ulcer diverticula occur in this area. Lesions at the apex of the bulb may be of either type, and differentiation may be impossible until response to treatment has been observed. For practical purposes, neoplastic ulceration need not be considered in the differential diagnosis of lesions in the bulb. It does require consideration in those of the second, third, and fourth portions. The roentgenologic manifestations are usually clear cut and associated with loss of appetite, weight loss, and occult blood in the feces.

Duodenal diverticula rarely become inflamed, probably because of the sterility of their contents, their retroperitoneal position, and their inverted position and wide ostia. Perforation is a rare sequela. The advisability of surgical treatment in most duodenal diverticula is highly questionable.

Diverticula are found less frequently in the jejunum than in any other portion of the gastro-intestinal tract and are usually discovered accidentally at operation, autopsy, or by roentgen ray. When uncomplicated, they are rarely if ever responsible for symptoms and require no treatment.

Meckel's diverticulum occurs as a result of incomplete obliteration of the omphalomesenteric duct, its structure depending on the degree of obliteration. The diverticulum is usually situated on the antimesenteric side of the ileum, 30 to 90 cm. proximal to the ileocecal valve. It may be attached to other viscera, or to the abdominal wall; rarely it is located between the leaves of the mesentery. The size of the opening into the ileum is important, for if the opening is wide, it permits the unhindered entrance and exit of intestinal contents. However, this wide neck may also permit the lodgement of a large variety of foreign bodies. The symptoms depend primarily on the nature of the complication present, obstruction and inflammation being most frequent in adults, peptic ulcer with hemorrhage in children. The acute catarrhal, phlegmonous, and gangrenous forms of diverticulitis occur, complicated by perforation with abscess formation or peritonitis. The symptoms so simulate those of acute appendicitis that a preoperative diagnosis is rarely made, although a periumbilical location of pain and tenderness is suggestive of diverticulitis.

Diverticular of the colon, as in other parts of the digestive tract, have been classified as true or false, congenital or acquired. Congenital true diverticula involving all layers of the intestinal wall are seen occasionally. The vast majority of colonic diverticula are formed by the herniation of the mucosa through the muscular layers. Clinical manifestations appear only when secondary inflammation develops, due chiefly to fecal obstruction of the neck, which prevents ade-

quate drainage and leads to pressure necrosis. In some cases diverticulitis may produce a stony-hard fixed mass quite indistinguishable on rectal examination from inoperable carcinoma. Roentgen differentiation depends on the fact that carcinoma tends to destroy the mucosa and produce a margin in which no mucosal pattern can be seen, while in diverticulitis the mucosal patterns persist and are usually exaggerated.

In summary, diverticula of the digestive tract, particularly of the colon, are extremely common; the esophagus and duodenum are quite frequently affected; the stomach and jejunum rarely so. Uncomplicated diverticula do not as a rule produce symptoms. Diverticula of the esophagus proper, of the stomach, duodenum, and small intestine rarely become inflamed, except for Meckel's diverticula. Diverticula may produce intestinal obstruction from intussusception or from adhesions. Illustrative case reports, are included.

STEPHEN N. TAGER, M.D.

**Study of the Mucosal Relief in Early Cancer of the Stomach with the Aid of Pharmaco-radiography.** M. Guy Albot and Guillemette Marquis. *Semaine d. hôp. Paris* (Special Number), September 1947, pp. 111-124.

The authors have succeeded in making a very early diagnosis of cancer of the gastric mucosa in 6 cases by combining radiology with the administration of morphine, insulin, or ipecacuanha. These drugs provoke hypertonic contractions of the muscularis mucosae, with the result that abnormalities in the gastric image are more strikingly demonstrated. Excellent roentgenograms bearing out the statements in the text are reproduced, and photographs and photomicrographs of the operative specimens are also furnished. Unfortunately details as to the dosage of the drugs and the exact time of administration in relation to the radiological examination are not given.

A considerable number of reports on this technic have appeared in the recent French literature. Among the references in the authors' extensive bibliography are several other papers by Albot and his associates, notably *Arch. d. mal. de l'app. digestif* 34: 139, 1945; 35: 189, 244, 1946.

DR. LEONARDO GUZMAN

**Pernicious Anemia and Susceptibility to Gastric Neoplasms.** Henry S. Kaplan and Leo G. Rigler. *J. Lab. & Clin. Med.* 32: 644-653, June 1947.

This article represents an extensive review of the literature on the relation of pernicious anemia to gastric neoplasms. Statistical studies show that the incidence of carcinoma of the stomach is much higher among patients with pernicious anemia than the expected incidence in a population of comparable age. Gastric polyps are also found with increased frequency in association with pernicious anemia. It thus appears that some etiologic relationship must exist between the two conditions. Three possibilities are considered: (1) that pernicious anemia directly produces a precancerous state of the stomach; (2) that gastric cancer causes pernicious anemia; (3) that the two diseases are linked through a precursor or manifestation common to both. The last of these seems the most likely. The authors point out that there are familial tendencies in both diseases, indicative of a common constitutional or hereditary factor that may well link the etiologies.

An interesting point brought out is the fact that the number of reports of coexisting pernicious anemia and

gastric cancer has increased since the advent of liver therapy. This has been interpreted by some to mean that the longer survival of the patients with anemia permits the development of cancer in a greater number. It is also speculated that liver extract itself may contain a weak carcinogenic agent which on prolonged administration may produce cancer in a susceptible individual, and experimental studies lend some support to this view.

The paper concludes with a section on the early detection of gastric cancer. The most obvious way to attack this problem is to examine routinely by roentgenologic or gastroscopic methods, or both, all persons in the gastric-cancer-bearing age. The expenditure of time and effort in such non-selective surveys, however, has been shown to be far out of proportion to the very small number of tumors discovered. Some basis of selection of patients more liable to gastric cancer than the general population is necessary. The only known "indicator" of this kind is pernicious anemia. Other possible indicators, as achlorhydria, chronic gastritis, and gall-bladder disease, are now being studied as a necessary prerequisite to the establishment of practical selective mass surveys for the early detection of neoplasms of the stomach.

SYDNEY F. THOMAS, M.D.

**Prolapse of Redundant or Hypertrophied Gastric Mucosa.** S. P. Bralow and M. Melamed. *Am. J. Digest. Dis.* 14: 215-221, July 1947.

It is the authors' contention that prolapse of gastric mucosa into the duodenum occurs often enough to warrant study, even though Bockus (*Gastroenterology* 7: 762, 1946) believes that the condition is so rare as to have no practical significance and Alvarez regards it as usually of no importance.

The etiology of the condition is unknown. One theory is that low-grade inflammation may produce hypertrophied rugae that are pushed into the duodenal cap by peristaltic waves. Another theory presupposes a narrowed pyloric lumen. Hyperperistalsis ensues in an attempt to force the gastric contents through the small opening, the attachment of the mucous membrane to the muscularis is loosened, the mobilized mucosa is subject to trauma, and hypertrophy and prolapse result. Neither of these theories has been substantiated at operation, for when surgical exploration has been done, the prolapsed mucous membrane has usually appeared normal and no evidence of a narrowed pyloric canal has been obtained.

A prolapse of gastric mucosa into the duodenum should be suspected in patients with an atypical ulcer history or those who are said to have functional complaints. The following points are of significance for the roentgen diagnosis:

1. The filling defect is located in the base immediately around the pyloric opening.
2. It will vary in size on repeated examinations and may even become temporarily reduced.
3. The rugae can be traced through the pylorus into the cap.
4. The bulb is not irritable, and no ulcer craters are seen.
5. Gastric peristalsis is more active than in the average patient.

The authors give 4 case histories with excellent drawings and film reproductions of the duodenal cap.

JOSEPH T. DANZER, M.D.

**A Study of the Gastric Stoma After Partial Gastrectomy: An Analysis of Ninety Gastric Resections.** Charles S. Kennedy, Roland P. Reynolds, and Meyer O. Cantor. *Surgery* 22: 41-47, July 1947.

The authors present their conception of what constitutes the true gastric stoma following gastric resection with gastro-enterostomy. Most surgeons have felt that the gastric stoma is represented by the communication between the stump of the stomach and the jejunum to which it is sutured. By means of diagrams and roentgenograms of living patients and autopsy specimens, it is shown that the portion of the jejunum sutured to the distal end of the stump of the stomach functionally becomes incorporated as part of the stomach. This means that the true gastric stoma is just distal to the point where the greater curvature of the stomach meets the jejunal wall. The portion of the jejunum sutured to the stomach dilates, as does the portion just distal to the anastomosis, narrowing like a funnel to a point where the jejunum of normal caliber is reached. This point is taken by the authors as being the real gastric stoma, which determines the rate of emptying of the stomach. They point out that if the actual anastomosis were considered the stoma, edema out of all proportion to what is known to occur would have to be present to cause the gastric retention that invariably occurs in the first week following gastro-enterostomy. The size of the true gastric stoma in the jejunum remains the same regardless of the type of gastric operation done. Surgeons are cautioned in bringing up the jejunal loop to the stomach to see that no twisting occurs distal to the anastomosis, as this would interfere with gastric emptying.

J. E. WHITELEATHER, M.D.

**Tertiary Gastric Syphilis.** William A. Knight and Abraham Falk. *Gastroenterology* 9: 17-27, July 1947.

The majority of reported cases of gastric syphilis represent the tertiary phases of the disease. Grossly and histologically, these fall into four groups: gummatous hyperplasia, ulcer, gastritis, and diffuse fibrosis (linitis plastica). The most characteristic gastric lesions are the shallow ulceration not extending below the submucosa; the diffuse inflammatory changes of the submucosa characterized by edema, cellular infiltration and diffuse granulation tissue; and the perivascular changes of proliferating endarteritis and panphlebitis.

The case of tertiary gastric syphilis reported in this paper had the pathological features enumerated above as well as the essential clinical criteria established by Bockus, namely, evidence of untreated tertiary syphilis; a demonstrable roentgen defect; the presence of gastric symptoms; the inability to alleviate symptoms or effect an improvement in the anatomic x-ray defect by orthodox methods of management without antisyphilitic treatment; symptomatic relief and disappearance of the x-ray defect after intensive specific therapy. The initial clinical manifestation was gastric hemorrhage. Subsequent normal gastro-intestinal x-ray series antedated the later confirmed syphilitic lesion by approximately two years. The etiology of the hemorrhage was unknown, but bleeding from syphilitic gastritis does occur. The excellent and early response to intensive therapy was attributed to the addition of penicillin to antisyphilitic treatment.

Evaluation of the multiple gastroscopic findings elicited in this case and those described by others lend support to the contention of Palmer, Schindler *et al.* (Ann. Int. Med. 18: 393, 1943) that there are insufficient reports of gastroscopic findings in gastric syphilis upon which to establish a definite diagnosis.

M. WENDELL DIETZ, M.D.

**Congenital Diaphragm of the Duodenum. Case Report with Preoperative X-Ray Studies.** Wallace I. Nelson. Minnesota Med. 30: 745-752, July 1947.

Congenital diaphragm of the duodenum is a developmental anomaly in which a membrane, formed by an infolding of the mucosa and submucosa, extends across the lumen of the duodenum. It is to be differentiated from stenosis and atresia. Some of the diaphragms present an aperture; others are intact. The literature contains reports of about 35 cases. In only 6 of these was the diagnosis made during life. The membrane varies in thickness from 0.5 to 4 mm. At operation a ring of constriction may be visible.

In cases of complete diaphragm, obstructive symptoms are prominent from the time of birth, in contrast to congenital pyloric stenosis, which is more apt to become manifest about the third week of life; in the latter condition, the vomitus does not contain bile. The important conditions in differential diagnosis are abnormal fixation of the duodenum, persistence of the hepato-duodenal ligament, annular pancreas, and vascular anomalies, extrinsically, and atresia and stenosis, intrinsically.

The operative technic is discussed and a case is reported in a twenty-six-year-old adult with obstructive symptoms due to an incomplete diaphragm.

PERCY J. DELANO, M.D.

**Congenital Atresia of Intestine and Colon.** Willis J. Potts. Surg., Gynec. & Obst. 85: 14-19, July 1947.

The prognosis for newborn infants with congenital atresia of the bowel has improved considerably in the past twenty years due to earlier diagnosis, chemotherapy, and improved operative technic.

Because of the infrequency of congenital atresia the diagnosis is often not made until the symptoms of bowel obstruction occur. Vomiting is the outstanding early symptom, occurring on the first or second day, depending on the intestinal level of the obstruction. The level also affects the degree of distention, which is greater the lower the obstruction. The stool is small, light green, and mucoid rather than having the usual tarry appearance of meconium.

The roentgen examination is important and should be done with the infant upright. The distended bowel loops due to obstruction are seen. The exact level of obstruction, however, may be impossible to determine because the greatly dilated loops are often crowded into abnormal positions. Barium by mouth is condemned. Treatment consists, briefly, in deflation of the bowel above the site of atresia by aspiration and a side-to-side anastomosis between the proximal and distal loops.

Five cases with one fatality are reported. In one case the site of the atresia was the transverse colon. This patient is believed to be the first with atresia of the transverse colon to survive operation.

ARTHUR W. PRYDE, M.D.

**Volvulus of the Cecum and Ascending Colon.** Edward L. Young, Harvey R. Morrison, and Walter E. Wilson, Jr. New England J. Med. 237: 78-86, July 17, 1947.

Volvulus of the cecum and ascending colon is certainly uncommon, but the diagnosis and treatment are quite specific and may determine the outcome of the disease. The occurrence of volvulus rests on the presence of a highly mobile cecum and ascending colon. The exciting cause may be violent peristalsis, abdominal tumor, mesenteric cyst, fecalith, foreign body, direct violence, constipation, acute appendicitis, or pregnancy.

The diagnosis depends on a careful x-ray study, the history, and physical findings. There is a definite history of intestinal obstruction with a sudden onset of acute abdominal pain centered in the lower right quadrant with nausea and vomiting. The physical findings are distention, tenderness, and abdominal spasm. Peristaltic activity may be demonstrated or there may be a quiet abdomen. Moderate leukocytosis is the rule.

A scout film of the abdomen may show a greatly dilated cecum and ascending colon with the terminal ileum. The dilated cecum is usually found in the upper left quadrant with complete lack of bowel markings in the lower right quadrant. These findings alone may suggest volvulus of the cecum. A barium enema will show a point of obstruction with twisting and torsion of the mucosal pattern.

Conditions to be differentiated are obstructing tumor, adhesions, dilated obstructed stomach, and redundancy of the colon. The twisting mucosal pattern is characteristic of volvulus. A small swallow of barium will outline the stomach and differentiate it from the distended cecum. The redundant colon may be outlined by barium.

Treatment of this condition is very important. If there is any question as to the viability of the proximal segment, a Mikulicz resection and exteriorization should be done. If the bowel has not been damaged, simple detorsion with fixation of the cecum may be sufficient.

Seven detailed case reports of volvulus of the cecum are presented, with reproductions of films showing the point of obstruction and the twisting pattern of the barium.

JOHN B. MCANENY, M.D.

**Diverticulitis of the Colon in Gynecology.** Edward Allen and L. Bruce Donaldson. West. J. Surg. 55: 393-400, July 1947.

The authors review 50 cases of diverticulitis of the colon seen and treated by the gynecological staff of their hospital in the past twelve years. Abdominal pain was present in forty-nine. During the acute phase of the disease, this pain was sharp and located in the left lower abdominal quadrant. There was an associated, tender, palpable sigmoidal mass in 12 cases. Other complaints were backache; abdominal distention, belching, and flatulence; constipation and diarrhea, and bladder disturbances. X-ray studies of the stomach and duodenum are indicated to rule out ulcer and gallbladder disease, and cystoscopy to exclude genitourinary conditions.

Many cases of diverticulitis have characteristic pelvic findings. There may be felt an indurated, nodular, lava-like tender mass, usually fixed high in the left pelvis. The inflamed sigmoid often pulls the uterus

to the left, although in some cases it is pushed away from the inflamed bowel. Movement of the uterus may produce pain. If there is chronic perforation of an infected diverticulum, a pelvic abscess may be palpable. Rectovaginal examination is very important. By means of the rectal finger, the examiner may be able to move the inflamed bowel separately from the adnexa. If there is adnexal disease, the pelvic mass may be moved independently of the bowel. Often the uterus can be outlined and separated from the bowel mass by this procedure.

Fifty-one pelvic examinations were recorded in this series. Fourteen were considered normal. In 10 instances a mass was felt which was thought to be due to diverticulitis. Other diagnoses included pelvic inflammatory disease, ovarian cyst, uterine fibroids, and tubal pregnancy.

Carcinoma of the bowel was diagnosed in 7 cases. The patients are in the same age group, but in cancer the course is persistently downhill, while diverticulitis is characterized by a chronic course with recurrent flare-ups.

In arriving at a diagnosis of diverticulitis, it is essential that the gynecologist bear in mind the possibility of the disease as a cause of abdominal and pelvic complaints in women past the age of thirty-five, particularly if the general clinical picture fits and/or if there is a mass in the left side of the abdomen and pelvis.

Colonic x-ray studies are the best single diagnostic facility. The detection of obstruction, point tenderness, the presence of diverticula, or a saw-tooth appearance of the involved segment with evidence of spasm, are all important points in diagnosis. In the 59 cases analyzed, initial bowel x-ray studies were done in 48, and a diagnosis of colonic diverticulitis was made in 42. Two of the 42 were negative on first examination, but on repeat examination showed the disease.

In 19 cases laparotomy was done. In only 4 of these was the true diagnosis suspected and these had intestinal obstruction; in the others, the preoperative diagnosis was in error. In 6 of the 19, bowel x-rays had not been ordered; in 7, x-ray studies missed the presence of diverticulitis. In 17, pelvic examinations had been made, and in every instance a mass was described which should have made the examiner suspicious of diverticulitis. Posterior colpotomy was performed for diagnosis in one case and for drainage of pelvic abscesses in four others. This measure is not advocated by the authors. Surgery or radium therapy in the presence of colonic diverticulitis may lead to adhesions, intestinal obstruction, fistulas, abscess formation, and peritonitis. If the patient is thoroughly studied, the diagnosis can be made and proper treatment instituted.

B. S. KALAYJIAN, M.D.

**Gallstone Ileus.** Ted Lacey. *Am. J. Surg.* 74: 86-89, July 1947.

A case of obstruction of the small intestine by a gallstone is reported, in which the stone is believed to have passed *via* the common duct and ampulla of Vater into the duodenum, and finally lodged in a kinked portion of the ileum, the kinking being due to adhesions from previous pelvic surgery. Because the condition is rare, accounting for only 1 to 2 per cent of all intestinal obstruction, an early diagnosis is frequently not made. Hence, a mortality of from 50 to 70 per cent is recorded in the literature, largely due to delayed treatment. The roentgenologist must keep this possibility in mind,

especially when an increased density is seen in the first roentgenogram associated with possible intestinal obstruction. A large shadow in the region of the right kidney pelvis was misinterpreted as a staghorn calculus in the case reported and the cause of the obstruction was not determined until an exploratory operation was done.

JOHN H. FREED, M.D.

**Experimental Studies on Biliary Regurgitation During Cholangiography.** Harry W. Mixer, Leo G. Rigler, and Miguel V. Gonzalez Oddone. *Gastroenterology* 9: 64-80, July 1947.

In their initial report (*Radiology* 48: 463, May 1947) on biliary regurgitation, the authors noted contrast in the kidneys at short intervals following cholangiography in clinical cases with common duct obstruction. In the present paper, experimental studies are described supporting the thesis that the dye reaches the kidneys after being regurgitated through the liver into the blood stream rather than by absorption through the biliary duct mucosa.

Thorotrast is a colloidal insoluble suspension whose presence in the blood stream can be demonstrated by radiographic and microscopic detection of its deposition in the reticuloendothelial system of the spleen. Animal experiments indicating thorotrast regurgitation through the liver into the blood stream preclude the possibility of absorption by the mucosa of the bile ducts because of its insolubility. For such regurgitation to take place, there must be rupture of bile capillaries to allow the colloid to pass into the blood and lymph directly.

Further studies on the dog proved that only very slight pressure is necessary to produce complete filling of the biliary tree. Excessive pressure is undesirable because of the possibility of forcing bacteria or other foreign material into the blood stream. All solutions used in cholangiography should be sterile. Special caution should be used in injecting patients with cholangitis or liver abscesses.

Studies employing radioactive phosphorus and the Geiger counter were undertaken because of the more sensitive method of detection inherent in this system. Findings indicated that minute quantities of  $P^{32}$  were present in both the blood and the lymph within five minutes after injection into the common bile duct. The lymph concentration was slightly but not significantly greater than that of the blood.

M. WENDELL DIETZ, M.D.

## THE MUSCULOSKELETAL SYSTEM

**Cranial Manifestations of Fibrous Dysplasia of Bone. Their Relation to Leontiasis Ossea and to Simple Bone Cysts of the Vault.** Frank Windholz. *Am. J. Roentgenol.* 58: 51-63, July 1947.

Occasionally skull changes are the first and only tangible evidence of fibrous dysplasia of the bone. These do not always correspond roentgenographically to changes elsewhere in the skeleton, where broadening of the bone, thinning of the cortex, and a rarefied, trabeculated appearance of the spongy structure give the impression of cyst formation. In contrast, there is a tendency in severe and advanced cases toward formation of sclerotic, dense bony deposits. This is most marked when the disease extends to the base of the skull, paranasal sinuses, or the nasal cavity. Such cases have been frequently described as leontiasis ossea.

In the disease, the thinning of the skull to the is of the skeleton. Occasional New-fel varium associated leontiasis. Fibrous childhood disease. Endocrines are present symptoms displacement narrow symptoms disease of normal and in average. The transformation into structural tion. Bone is transformed Radiolucent lamellar bone tissue. The radiolucent tissue rather than Prior to the lesion if localized hypertrophy osteitis advanced osseous. Polyostotic Girls. Int. Med. The aplasia (A) because twins, a under other report. The endocrine dysplasia, development pigmentary skeletal

In limited involvement of the skull, the foci of the disease appear as simple, rounded, cyst-like translucencies of the vault with widening of the diploe and thinning of the tables. These cysts may or may not protrude beyond the outer table of the skull. Where there is moderate cranial involvement, development of the disease usually dates from early childhood. The cranium and face may be seriously distorted. There is remarkable unilateral localization of abnormalities of the skull and facial bones. This is usually homologous to the side of the skeletal involvement, a finding which is of diagnostic value. In extreme involvement of the skeleton, skull changes are correspondingly serious. Occasionally the whole skull may become involved. New-formed bone masses may be deposited on the calvarium and base of the skull. Bone deformities are associated with marked changes in facial appearance—leontiasis ossea.

Fibrous dysplasia of bone is primarily a disease of childhood, though it is not encountered until adolescence or adult life. It is more frequent in females. Endocrine disturbances and cutaneous pigmentation are present in about one-third of the cases. Focal symptoms may occur from local cortical irritation and displacement of adjacent structures. There may be narrowing of the nerve canals and associated clinical symptoms as a result of pressure. Progress of the disease in the skull seems to stop with the termination of normal bone growth or with puberty. The calcium and inorganic phosphorus content of the blood is average.

The primary histopathological change seems to be the transformation of rather extensive areas of bone marrow into connective tissue, following severe bone destruction by osteoclastic activity and lacunar resorption. In the connective tissue, primitive non-lamellar bone is formed. This may remain uncalcified or be transformed into calcified irregular lamellar bone. Radiolucent areas are the result of replacement of lamellar bone by connective, osteoid, and non-lamellar bone tissues. This is the basis of cyst formation. The radiopaque areas are newly formed calcified osseous tissue due to metaplastic activity in connective tissue rather than to osteoblastic activity.

Prior to the segregation of fibrous dysplasia from related conditions, misinterpretations were frequent. The lesions were classified as cranial hyperostosis or, if localization was unilateral, as hemicraniosis, hemihypertrophy, unilateral von Recklinghausen's disease, osteitis fibrosa cystica of the skull, etc. Most of the advanced cases, however, were reported as leontiasis ossea.

CLARENCE E. WEAVER, M.D.

**Polyostotic Fibrous Dysplasia in One of Negro Twin Girls.** Guy A. Caldwell and T. F. Broderick, Jr. *Ann. Int. Med.* 27: 114-126, July 1947.

The authors report a case of polyostotic fibrous dysplasia (Albright's syndrome) which is of special interest because of its occurrence in one of proved dizygotic twins, a Negro girl of nine years. The child had been under observation for twenty months at the time of the report.

The most striking features of this case are obvious endocrine dysfunction, generalized but asymmetrical dysplasia of the skeleton, premature physical development and epiphyseal closure, and patchy skin pigmentation having little apparent relation to the skeletal lesions. Blood calcium and phosphorus were

repeatedly found to be normal, although the alkaline phosphatase was consistently elevated. There was no evidence to support the assumption that heredity, environment, or infection were etiologic factors. A primary germ plasm defect seems to be the most plausible explanation.

STEPHEN N. TAGER, M.D.

**Alkaptonuric Arthritis: Cause for Free Intra-articular Bodies.** C. J. Sutro and M. E. Anderson. *Surgery* 22: 120-124, July 1947.

Alkaptonuria results from incomplete catabolism of the proteins tyrosine and phenylalanine, with the formation of an intermediate product, homogentisic acid, an alkapton body which is deposited in the tissues and eliminated in the urine, causing it to turn dark brown on exposure to air. Deposition of these alkapton bodies or pigmented derivatives takes place for the most part in relatively avascular tissues or in those with poor metabolism, resulting in ochronosis. Musculoskeletal tissues, when infiltrated with these pigments, may undergo calcification, ossification, or degenerative changes commonly seen in osteoarthritis. Free bodies may form in large joints thus involved.

In the case reported, roentgenograms of the spine showed narrowing and calcification of the intervertebral disk regions of the dorsal and lumbar vertebrae. The interpubic ligaments were also calcified. Radiographs of the knees showed a marked irregularity and sclerosis in the subchondral region of the left patella and of the contiguous femoral articular surface. Small osteophytes were present at the periphery of the articular surfaces of the tibia and femur in both knees. Several free roundish bodies were noted in the posterior compartment of the left knee. No free bodies were present in the right knee joint.

It is believed that articular cartilage prematurely degenerates when homogentisic acid is deposited in it. Such cartilage becomes friable, is easily cracked, and is separated readily from the subchondral zone. Detached cartilage fragments may be deposited on the synovial lining or lie free in the joint cavity. A secondary disturbance in the metabolism and nutrition of the cartilages may occur, leading to further degeneration and calcification. These detached fragments of cartilage form the nidus for the free bodies found associated with alkaptonuria and ochronosis. The articular cartilage damage may lead to premature secondary generalized osteoarthritis. Pigment in the villi of the joint may lead to proliferative villous synovitis.

Operation was not done in the case reported, the presumptive diagnosis being made on the basis of chemical findings in the urine associated with the radiographic change in the skeleton. Conditions to be differentiated are melanuria, chronic argyria, methemoglobinemia, hemochromatosis, porphyria, carotinemia, Gaucher's disease, chloroma, neurofibromatosis, and fibrous dysplasia.

J. E. WHITELEATHER, M.D.

**Calcified Medullary Defects in Bone.** Albert Barnett Ferguson, Jr. *J. Bone & Joint Surg.* 29: 598-602, July 1947.

Calcified medullary defects in bone are usually solitary, symptomless, and somewhat rounded in shape. They are found in or near the metaphyseal region of the long bones. The lesion is usually in the distal femur, less frequently in the proximal tibia, femur, and humerus. The patients do not complain of pain and the

lesion is discovered during radiography for some other cause. Differentiation must be made from caisson disease of bone, chondrodysplasia, osteoid osteoma, sclerosing osteogenic sarcoma, osteoplastic prostatic metastases, and bone infarct.

The case reported is that of a sixty-eight-year-old male with a fracture in the distal femur, where a calcified medullary defect was noted in the distal fragment. At operation, this focus was removed and found to be non-viable cartilage, with a slow process of new bone formation about the periphery.

It is believed that this lesion is a degenerated calcified island of cartilage which did not progress to complete ossification at the time of skeletal growth.

JOHN B. McANENY, M.D.

**Osteoid Osteoma.** Ignacio Ponseti and Chester K. Barta. *J. Bone & Joint Surg.* 29: 767-776, July 1947.

Seven cases of osteoid osteoma have been found by the authors, the lesion occurring in a rib, in the calcaneus, in the proximal tibia, the middle phalanx of the index finger, the lower half of the fibula, and in the left ilium. Three patients were males and four were females, with the ages ranging between fourteen months and twelve years. There is a history of pain of slow onset, and sometimes intermittent in character, not relieved by rest but present for a long period of time. There is usually marked tenderness with limitation of motion and muscle spasm when the lesion is near a joint (Sherman: *J. Bone & Joint Surg.* 29: 483, 1947. Abst. in *Radiology* 50: 269, 1948). The patient with the lesion in the rib showed a definite curvature of the spine; the patient with the lesion in the lower fibula showed a deformity of the ankle; and the patient with the lesion in the upper tibia showed a bowing of that bone.

Osteoid osteoma appears on roentgenograms as a circumscribed area from 0.5 to 2.5 cm. in diameter, usually uniform but sometimes mottled in appearance. It is surrounded in most cases by reactive new bone formation. No evidence of infection is found in any of these lesions and the authors believe, therefore, along with Jaffe, that the lesion is a benign neoplasm rather than an infectious process.

This group of cases suggests that the best treatment of osteoid osteoma is an extensive block dissection rather than curettage, following which the lesion may recur several times.

The seven case reports are included, along with numerous reproductions of roentgenograms.

JOHN B. McANENY, M.D.

**Eosinophilic Granuloma of Bone.** Robert N. Cooley and Glenn D. Carlson. *Texas State J. Med.* 43: 64-69, June 1947.

Eight cases of eosinophilic granuloma of bone are reviewed and a brief review of the literature is given. Four of the cases are well illustrated.

SYDNEY F. THOMAS, M.D.

**Cooley's Anemia (Mediterranean Anemia) in a Chinese Child.** Annie V. Scott. *Chinese M. J.* 65: 77-84, March-April 1947.

A detailed report of a case of Cooley's anemia in an American-born Chinese child is presented. So far it has not been determined whether this disease is the result of peculiarities in the hematopoietic system or of metabolic abnormalities. The onset is early in infancy

with signs of anemia and splenic enlargement. Changes in the orbital and malar bones, together with the muddy coloration of the skin often produce a peculiar mongoloid facies in children of Italian or Greek parentage, among whom the condition was first reported. The characteristic blood findings are excessive destruction by fragmentation of the red cells and a peculiar irregular deposition of hemoglobin in the cells.

The x-ray findings consist of widening and increased transparency of the long bone shafts, with thinning of the cortices due to pressure from a hyperplastic bone marrow. Medullary trabeculae are prominent. In the later stages, the skull shows similar changes, with the outer table becoming extremely thin. Vertical bone striations, perpendicular to the plane of the inner table, provide a characteristic roentgen feature. In the case reported the changes were particularly conspicuous in the distal ends of the femurs, where the lateral margins were convex rather than concave and a coarse reticular pattern was demonstrable. There was distinct thickening of the inferior half of the vertical plate of the frontal bone and the nasofrontal process. In the superior segments of the parietal bones there was an irregular rarefaction suggesting unusual prominence of the diploic spaces. The bones of the thoracic cage showed changes similar to those in the long bones.

Medical treatment, consisting of iron, bone marrow, and liver, administered *via* various routes, is ineffective. Splenectomy is a palliative measure, and transfusions help prolong life, but do not reverse the anemic process.

LOUIS BERNSTEIN, M.D.

**Roentgenographic Features of Metastases of a Retinoblastoma to the Long Bones.** Report of a Case. Ralph E. Rowen. *J. Bone & Joint Surg.* 29: 805-808, July 1947.

The roentgenographic appearance of metastases from a retinoblastoma is not very well known and is poorly represented in the literature. The author reports the case of a Negro boy of six who had his left eye removed for a retinoblastoma and later had a swelling of the lower end of the right arm. Roentgenograms showed atrophy of the distal right humerus with soft-tissue swelling and vertical striations of the periosteum, with irregular areas of bone absorption. There was new bone formation and the appearance suggested an osteogenic sarcoma. The proximal end of the left humerus also showed the same type of periosteal new bone formation with vertical periosteal striations. The skull showed demineralization of the parietal bones. There was no evidence of metastasis in the chest.

In the differential diagnosis of osteogenic sarcoma, one must consider metastases from a retinoblastoma which, however, does not appear to show any characteristic roentgen change.

JOHN B. McANENY, M.D.

**Ankylosing (Marie-Strümpell) Spondylitis (Analysis of 100 Cases).** Wallace Graham and M. A. Ogryzlo. *Canad. M. A. J.* 57: 16-21, July 1947.

This article concerns a group of 100 cases of Marie-Strümpell disease reported from the Joint Services Arthritis Center at St. Thomas, Ontario. Few cases were recognized early, when prompt institution of therapy would have been most beneficial. In this series an average of five and seven-tenths years elapsed

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from the time of onset of symptoms to the recognition of the disease. It would seem of value, therefore, to present a study particularly of the early features which, in retrospect, reveal a somewhat uniform clinical pattern which has not been sufficiently recognized or given the clinical prominence which it deserves.

Ankylosing spondylitis is by no means a medical curiosity. In a recent study by Boland and Present (J. A. M. A. 129: 843, 1945) 18 per cent of patients admitted to a U. S. Army General Hospital, with chronic back complaints, were found to have this disease. At this Arthritis Center the ratio was 1 case of spondylitis to every 2.8 cases of rheumatoid arthritis. In 73 per cent of the cases in this series the onset of the disease occurred before the age of thirty, the average age being twenty-four years.

The possible precipitating factors included trauma, exposure, and urethral infection, but in the majority of cases no precipitating factor was elicited.

In a search for symptoms the authors found that 82 per cent of their patients complained of back pain, 12 per cent had symptoms referable to the peripheral joints, and 6 per cent showed chest symptoms.

In the present series the sacroiliac joints were involved in all cases and there was calcification in the paravertebral ligaments in 42 cases. A variable degree of osteoporosis was observed, and in a few cases this was a striking feature, with marked bulging of the intervertebral disks and central narrowing of the adjacent vertebral bodies.

The authors concluded that ankylosing spondylitis should be suspected in young adult males complaining of stiffness and back pain with reduced spinal mobility and an increased erythrocyte sedimentation rate. The finding of bilateral sacroiliac arthritis on x-ray examination confirms the diagnosis.

HUGH A. O'NEILL, M.D.

**Variations in the Syndrome of the Ruptured Intervertebral Disc in the Lumbar Region.** Frederic V. Kristoff and Guy L. Odom. *Surgery* 22: 83-93, July 1947.

Approximately 80 per cent of cases of ruptured intervertebral disc present a fairly typical picture which has been well described in the literature. About 20 per cent are atypical and require careful differential diagnosis. These atypical cases may be roughly classified as follows:

1. Rupture of a disk at some space above the 4th lumbar. Complaints are mostly of back and flank pain without sciatic radiation or signs of lower disk rupture.

2. Absence of lateral protrusion or occurrence of protrusion large enough to affect more than one or two nerve roots. In such cases the syndrome may begin with low back pain progressing to a picture indicating cauda equina tumor or there may be signs and symptoms of compression of a single root becoming bilateral in character. Symptoms are noticeably changeable. Neuropathologic signs are vague and late.

3. Bilateral protrusion with bilateral sciatic radiation.

4. Multiple ruptures. These may be unilateral or bilateral and present a complex pattern.

5. Detachment of a ruptured fragment, which may settle anywhere in the canal and may shift around, causing variable symptoms and findings.

6. Very large rupture, producing a complete spinal block and compression of the cauda equina with signs

suggesting a tumor of the cauda equina. These large ruptures are associated with loss of bladder control, bowel control, and sexual potency. Rapid development of symptoms and signs following trauma is suggestive of disk rather than tumor.

For the diagnosis of these atypical cases careful correlation of clinical and operative findings is important. Myelography is of special value for the diagnosis of multiple ruptures, ruptures above the fourth interspace, cases without lateral protrusion, and wandering disk.

J. E. WHITELEATHER, M.D.

**Osteitis Pubis.** Norborne B. Powell. J. Bone & Joint Surg. 29: 785-787, July 1947.

Osteitis pubis is an infrequent complication of suprapubic prostatectomy, consisting in a periosteal reaction of the pubic bones. The onset may be from ten days to two months after operation, with a dull aching pain in the region of the symphysis pubis, extending along the inferior or superior pubic rami. The pain may radiate to the perineal region and inner aspects of the thighs. The patient is unable to walk and attempted motion of the lower extremities is extremely painful. This reaction may last from six weeks to two years.

The roentgen changes consist in roughening or fraying of the periosteum and a moth-eaten appearance of the symphysis pubis and rami evident about three weeks after operation. The disease is self-limiting, and recalcification of the bone usually occurs in six to ten weeks.

There is a possibility of confusing this condition with infection of the space of Retzius or of the perivesical spaces. The roentgenogram will identify the cause of disability.

A case report is presented of a patient who had a suprapubic cystectomy on Oct. 14, 1943. On Dec. 2, he was readmitted complaining of severe pain over the symphysis, and roentgenograms showed an osteitis of the pubic bones. Recovery followed symptomatic treatment, and roentgenograms of Jan. 20, 1944 showed no evidence of change in the bone.

JOHN B. McANENY, M.D.

**Shoulder and Elbow Lesions Distinctive of Baseball Players.** George E. Bennett. *Ann. Surg.* 126: 107-110, July 1947.

There are two types of lesions found in shoulders of baseball players. The first is distinctive and consists of deposits or exostoses along the posterior inferior rim of the glenoid fossa, produced by the abnormal strain exerted on the triceps in throwing a ball. The second lesion, seen also in association with other occupations, consists in fraying of the supraspinatus tendon. Neither of the lesions responds to surgery. X-ray demonstration of the lesions along the posterior glenoid rim is best accomplished with the patient lying supine and the arm elevated to shoulder level and rotated externally. The central beam is centered on the shoulder with 5 degrees angulation toward the head.

Pathologic lesions of the elbow joint—loose bodies and osteoarthritis—occur in ball players just as they do in non-ball players. Two distinctive lesions are, however, found. The sharp supination of the wrist, plus extension of the elbow in throwing a curved ball, leads to irritation of the tissues in front of the internal humeral condyle. Examination will reveal fullness over the pronator radii teres, with negative x-ray find-

ings. On rare occasions, lipping of the ulna at its articulation with the internal condyle is seen. The most distinctive lesion is the development of extra-articular, single or multiple deposits of bone in the ligamentous and tendinous attachments beneath the ulnar nerve. Removal of these deposits will generally produce relief.

LOUIS BERNSTEIN, M.D.

**Bilateral Congenital Fusion of the Semilunar and Cuneiform Bones. Report of a Case.** J. C. R. Hindenach. *Brit. J. Surg.* 35: 104-105, July 1947.

A brief report of a somewhat unusual anomaly. Complete fusion of the semilunar and cuneiform bones of both wrists was discovered incidentally during an x-ray examination following a minor football injury.

**Experiences with Epiphyseal Arrest in Correcting Discrepancies in Length of the Lower Extremities in Infantile Paralysis. A Method of Predicting the Effect.** William T. Green and Margaret Anderson. *J. Bone & Joint Surg.* 29: 659-675, July 1947.

In determining the time for epiphyseal arrest of growth in the lower extremities, the authors have been rather dissatisfied with the usual methods and have evolved one of their own. The method of prediction is based upon the skeletal age rather than the age of the patient in years. Orthoroentgenograms of the lower extremities were made at frequent intervals to determine the rate and amount of growth at the proximal tibial epiphysis. Charts and prediction tables have been arranged to determine the expected amount of growth in the proximal tibial epiphysis at a given age. Since very little growth occurs in the last year before the epiphysis closes, the bone growth is terminated at fourteen years three months in girls and sixteen years three months in boys.

These tables have been shown to compare favorably with other methods of prediction and have worked out very satisfactorily clinically. The authors expect to continue compiling data both in the subjects already in use and in new individuals. They caution about the irregularity in the growth pattern of an individual and suggest that this be considered as a limiting factor in judging any method of predicting epiphyseal line closure.

JOHN B. McANENY, M.D.

**Air Arthrography of the Knee Joint.** J. G. Bonnin and J. L. Boldero. *Surg., Gynec. & Obst.* 85: 64-70, July 1947.

By means of roentgenograms made after distention of the knee joint with air, the authors have been able to diagnose tears, ruptures, displacements, unusual thickening, and cysts of the semilunar cartilages. The cartilaginous layer on the condyles is also rendered visible, and injuries to this structure may be visualized.

Air studies were considered of particular value in: (1) cases in which no adequate history was available (many of the patients in this series did not speak English); (2) cases in which the clinical findings were inconclusive, both as to site of the lesion and its actual presence; (3) cases in which a double lesion was suspected, either a double tear, or a cyst and tear; (4) cases in which ligamentous laxity might account for the symptoms, and it was required to prove or disprove associated cartilage damage.

Of 75 cases studied, 49 came to operation; in 44 of these the roentgen findings were confirmed operatively.

Of the 5 cases missed, 3 were diagnosed as negative and very small tears were found at operation; in the remaining 2, tears were diagnosed but only hypertrophied fat pads were found. It is felt that if surgery is limited to the roentgenologically positive cases 100 per cent accuracy could be obtained. However, the small tears and the hypertrophied fat pads would thus be missed.

Judging by the excellent illustrations, the authors seem to have developed a superior technic. With horizontal x-ray beam, multiple tangential views are made through the joint space, which is separated slightly by forced abduction or adduction.

The technic is outlined in detail and illustrations, diagrams, and descriptions of the normal and abnormal are included.

ALFRED O. MILLER, M.D.

**Unclassified Premature Cessation of Epiphyseal Growth about the Knee Joint.** Otto C. Kestler. *J. Bone & Joint Surg.* 29: 788-797, July 1947.

It has been known that following tuberculosis premature arrest of growth of the epiphysis about the knee joint will occur. The author has found this condition to follow other types of infection and injuries, and to occur apparently without any predisposing cause.

One group of lesions includes infections about the hip joint, showing early arrest at the knee epiphysis. There is shortening of the entire extremity but with continued growth of the proximal fibular epiphysis. This gives a lengthening of the fibula over the length of the tibia with the proximal epiphyseal plate of the tibia being wider and usually slanting medially and downward. In the epiphyseal plate there is usually a sclerotic structure with a star-shaped area in its center. A second group consists of cases in which the hip disease is non-infectious, such as congenital dislocation. In these, similar changes of the knee are observed. In a third group are cases in which no apparent etiological agent is active; possibly the cause has disappeared without leaving trace in the bone structure. The author presents four complete case histories defining the hip lesions with the subsequent change at the knee. These changes at the knee almost invariably lead to a varus deformity.

In all, the author has seen 22 cases, the majority following infections of the hip joint. He believes that the hip lesion interferes somewhat with the blood supply in the lower extremity and causes disturbance of growth at the knee epiphysis, usually with the exception of the proximal fibular epiphysis. He believes that the changes at the knee begin six to eight months after the growth center at the hip is affected by the disease. Several years may elapse, however, before definite growth discrepancy is seen.

JOHN B. McANENY, M.D.

**Oblique Subcervical (Reverse Intertrochanteric) Fractures of the Femur.** Louis T. Wright. *J. Bone & Joint Surg.* 29: 707-710, July 1947.

The oblique subcervical fracture of the femur does not appear to have been described in the literature and is definitely uncommon. It is the reverse of an intertrochanteric fracture, with the fracture line extending parallel with the long axis of the femoral neck and cutting across the intertrochanteric region almost on a line with the lower border of the femoral neck. The importance in recognizing this type of fracture lies in its

physio-anatomical factors: (1) the fracture line is between the two limbs of the iliofemoral ligament; (2) the muscular attachments to the greater trochanter do not influence the movements or position of the distal fragment. The attachment of the lower portion of the iliofemoral ligament and the iliopsoas to the lesser trochanter tends to draw the lower fragment upward and inward beneath the femoral neck.

The fracture is produced by indirect trauma with the thigh in hyperextension, usually in patients of advanced age with muscular and ligamentous weakness and bone brittleness.

Three case histories are presented in detail and numerous reproductions of roentgenograms are presented. The author comments on the constant formation of good callus in all fractures.

JOHN B. MCANENY, M.D.

**Osteochondritis Dissecans of the Talus.** R. Beverley Ray and Edward J. Coughlin, Jr. *J. Bone & Joint Surg.* 29: 697-706, July 1947.

In a period of four years the authors collected a series of 13 cases of osteochondritis dissecans of the talus involving 14 ankles. This seems to be an unusually large number in the short period of time, but some of these patients were in military service, which possibly explains the prevalence of the disease. The authors believe that trauma is probably the exciting cause.

Pain, tenderness, swelling, and effusion about the ankle are usual features. The onset frequently follows a mild injury but may also occur after repeated minor traumata. On examination, there is usually swelling and, in long standing cases, chronic edema and periarticular thickening. The tenderness is most characteristic and may persist long after other symptoms have subsided. Limitation of motion, locking, instability, and a palpable loose body may be found.

Roentgenographic examination is usually diagnostic. The lesion in this series was found most frequently in the superomedial border of the articular surface of the talus, but the superolateral border may also be involved.

Some patients have been treated conservatively and others have had the lesion removed surgically. Several case reports are presented.

The authors caution against overlooking the lesion, especially on the lateral border of the articular surface of the talus, where it may be overshadowed by the fibula but can be brought out in an oblique view.

JOHN B. MCANENY, M.D.

**Solitary Unicameral Cyst of the Os Calcis.** Charles C. Verstandig. *New England J. Med.* 237: 21-22, July 3, 1947.

A single case of solitary cyst of the calcaneus is added to the 12 cases already reported in the literature. The lesion was demonstrable roentgenographically as a sharply marginated pyramidal radiolucent area involving the anterior half of the os calcis, lying parallel with the long trabeculations of the posterior half of the bone.

**Stress Fracture of a Metatarsal in a Young Child.** Lewis D. Rutter. *Brit. M. J.* 2: 55, July 12, 1947.

A stress fracture of the metatarsal in a 4-year-old girl is reported. The diagnosis was based on the sudden onset of pain on walking, without trauma; swelling of the foot (no bruising) and tenderness of the affected metatarsal shaft; and the x-ray appearances.

## GYNECOLOGY AND OBSTETRICS

**Role of Nutrition in Pelvic Variations.** Herbert Thoms. *Am. J. Obst. & Gynec.* 54: 62-73, July 1947.

To be thoroughly appreciated this article will have to be read in its entirety but the following statements are offered by the author. (1) Roentgenography has revealed that the adult female pelvis is subject to considerable variation in its anteroposterior relationship. These variations have a definite clinical interest to the obstetrician, for they may have a pronounced effect upon the course of labor. (2) The anatomical texts describing the so-called "normal" female pelvis need revision, because the architecture usually described is that of the brachypellic or oval pelvis, which is present in only about one-third of adult women, according to the evidence presented in this paper. (3) In infancy and childhood the pelvis is essentially similar in both sexes as far as the anteroposterior relationships are concerned; until a time just preceding the puberal period growth tends to be symmetrical. At that time changes in the anteroposterior and transverse relationships take place. These changes are apparently the results of two major influences, sex hormonal and nutritional. It would seem at first appraisal that hormonal influence was the dominant factor because of the well known somatic and psychic phenomena associated with the puberal period. Nevertheless, wide variations in pelvic architecture are present in our adult population which are apparently unrelated to such phenomena. (4) Evidence is given indicating that nutritional changes during the puberal period also play a major role in these changes in anteroposterior and transverse pelvic relationships. This role seems to be associated closely with calcium and vitamin C requirements. (5) The evidence here presented emphasizes the need for further study of the nutritional requirements of the growing child, particularly during the puberal period. It also emphasizes the importance of using what knowledge of this subject we already have to the end that these nutritional needs be met in a manner compatible to the demands of normal growth.

HUGH A. O'NEILL, M.D.

**Primary Carcinoma of the Fallopian Tube.** Morton Vesell and Harry Schneider. *Am. J. Obst. & Gynec.* 54: 140-144, July 1947.

Of all the derivatives of the müllerian duct, the fallopian tube is least often the site of carcinoma. According to the literature, primary carcinoma of the tube is found in from 0.34 to 1.33 per cent of operations on diseased tubes. Between 1928 and 1939, 1 case was reported at Mount Sinai Hospital, New York, while at Beth Israel Hospital, New York, 4 cases were seen during the same interval.

The preoperative diagnosis of primary carcinoma of the fallopian tube is seldom made. Occasionally it has been suspected, solely on the sign of intermittent watery and serosanguineous vaginal discharge in women past the menopause. While this clinical feature is suggestive, it is by no means pathognomonic. As associated conditions are usually present, such as uterine fibroids, ovarian cysts, diseased adnexa, etc., it is almost impossible in most cases to single out carcinoma of the tube. However, it is felt that salpingography might be a decided aid in making a preoperative diagnosis, and therefore should be employed more frequently when the disease is suspected.

The authors present 4 cases.

The first patient was operated upon May 1, 1945, and a myomatous uterus and a thickened sausage-shaped right fallopian tube were found. A total abdominal hysterectomy and bilateral salpingo-oophorectomy were performed, as the tubal mass was considered malignant. Microscopic examination revealed an anaplastic solid papillary carcinoma. Despite post-operative deep x-ray therapy, the patient died on August 1, 1946.

In the second case operation was done on July 17, 1945. A right tubo-ovarian mass was found adherent to the cul-de-sac; the left tube was dilated, and the uterus was essentially normal. A total abdominal hysterectomy and bilateral salpingo-oophorectomy were performed under spinal anesthesia. Microscopic examination of the right tube revealed a necrotic adenocarcinoma, while the left tube showed an atrophic hydrosalpinx.

Neither of the other 2 cases was diagnosed preoperatively. In spite of radical operation and x-ray therapy, one patient died within a year. The other was well at the time of the report a year after operation, though she received no postoperative irradiation. In neither patient was a history of symptoms of hydrops tubae profuens obtained. The authors believe that with hysterosalpingography, a preoperative diagnosis might have been made in each of these cases, and they recommend more frequent utilization of that procedure in the presence of pelvic masses in women past the menopause.

The literature reveals only an occasional case of tubal carcinoma with five-year survival.

DANIEL WILNER, M.D.

**An Office Procedure for Hysterosalpingography.** A. P. Hudgins. *West. J. Surg.* 55: 407-413, July 1947.

The author describes an indwelling, cervical, screw-type, self-retained cannula with a ball-type valve and detachable handle for hysterosalpingography in the gynecologist's office. The larger end of the cone-shaped instrument has an opening which (after the handle has been removed) accepts the regular tip of a Luer syringe or syringe extension.

The value of hysterosalpingography as a diagnostic procedure and for therapeutic results in some instances is well known. The author believes that with this instrument the examination may be done with greater ease and less time loss and certain advantages to the patient, the gynecologist, and the radiologist. He proposes that the cannula be inserted and the oil injected into the uterus (backflow being prevented by the ball valve) by the gynecologist and the patient be sent to the radiologist for film study. Then the patient is to return to the gynecologist for removal of the cannula. The film is made thirty minutes after the oil is injected and the cannula removed unless there is evidence of obstruction of the tubes, in which case it is left in for twelve to twenty-four hours and another film is made. The thirty-minute period is used since it is believed that this allows ample time for uterine contractions to force oil out in the tubes in most cases.

[To the abstractor this method of doing uterosalpingography appears to be decidedly inferior to the standard procedure of injecting the oil into the uterus under

fluoroscopic control in the radiology department or office. He does not agree with the author that there is little danger in injecting blindly, as is suggested here. There are too many reports of oil entering the uterine venous plexus to warrant approval of such a method. The abstractor believes that this method involves considerable risk, is far less likely to produce satisfactory results, and cannot be less painful for the patient than the standard method with teamwork between the gynecologist and radiologist. Saving of money and time does not excuse the use of an inferior diagnostic method.]

B. S. KALAYJIAN, M.D.

## THE GENITO-URINARY SYSTEM

**Excretory Cystograms after Voiding.** James R. Dillon. *California Med.* 67: 17-22, July 1947.

Many pathological conditions of the upper urinary tract and bladder can be diagnosed by excretory urography, not only from morphological deviation, but particularly by a study of the peristaltic action of the calices, renal pelves, and ureters and the emptying capacity of the kidney and bladder.

Routine preparation of the patient includes abstinence from fluids and food following the evening meal the day preceding examination; a cathartic at nine that evening; an enema, if necessary, one hour before examination. A scout film is made prior to injection of the opaque medium. Following injection, films are obtained at three to five minutes, fifteen minutes, and twenty-five minutes. An upright film immediately after the twenty-five minute film and a film through the bladder region immediately after voiding complete the series.

The present report stresses the study of the cystogram made immediately after voiding, and numerous reproductions illustrate the findings. Opaque urine in the retrotrigonal pouch behind the hypertrophied interureteric ridge and in the anterior pouch of the vertex of the bladder indicates urethral obstruction, particularly a fibrous contracted bladder neck. In the male, it indicates also a median bar or early hypertrophy of the prostate. A filling defect in the floor of the bladder with superoposterior displacement of the ureters, is due to hypertrophy of the prostate or to a mass in that area. In women, visualization of bladder neck contraction is indicative of trabeculation, urethral polyps, or Hunner's ulcer. Defects due to retroperitoneal abscess and diverticula have also been observed. This study also provides an excellent method of observing the ureters at the bladder entrance.

MAURICE D. SACHS, M.D.

**Unilateral Absence of the Kidney with Rudimentary Ureteral Anlage.** H. U. Gloor. *Schweiz. med. Wchnschr.* 77: 672-674, June 28, 1947.

Unilateral absence of the kidney, although compatible with life, often brings the patient to a physician because the solitary kidney is especially susceptible to disease. The diagnosis is relatively easy if the trigone has developed asymmetrically with only a single ureteral orifice, but the author reports three cases in which there was a unilateral kidney with a normal trigone and a short ureteral stump on the abnormal side. In one of these patients an exploratory laparotomy confirmed the absence of the kidney. Such cases offer a more difficult diagnostic problem.

LEWIS G. JACOBS, M.D.

**Radiologic Signs of Benign and Malignant Renal Tumors.** R. Hickel. *J. de radiol. et d'électrol.* 28: 94-103, 1947.

This article begins with a schema in which it is proposed to illustrate the different sorts of renal filling defects. These are arbitrarily chosen and described; there is no correlation with the different sorts of renal neoplasms which the pathologist recognizes, nor with the signs we ordinarily rely upon to suggest the kind of tumor which may be encountered at operation.

The reproductions of films fall into two groups. The first group consists of cases with operative confirmation; those of the second are presented speculatively. Even in the group with confirmation, the diagnosis is expressed merely as "malignant tumor." The display might be more helpful if an attempt was made at more exact definition, as "tumor of renal pelvis," or "tumor of lower pole," or "hypernephroma" or "hypernephric carcinoma" or "embryonal sarcoma" or some other pathologic form, with due consideration of differences of opinion as to classification.

I have seen a renal neoplasm lying in the pelvis, which looked very much like a large round calculus, and have also seen forms resembling cysts with calcification diagnosed by the pathologist as "hypernephric carcinoma." These and many other variations, such as extensive calcification in a hypernephroma, have provided a valuable range finder in ruling out possibilities, but only because they tend to bear on the subject of differentiating benign from malignant pyelographic appearances. The present paper does not contribute significantly to such differentiation.

PERCY J. DELANO, M.D.

**Malignancy of the Kidney, Survey of 195 Cases.** Albert P. Graham. *J. Urol.* 58: 10-21, July 1947.

This paper reviews all of the malignant neoplasms of the kidney seen at Edward J. Hines Memorial Hospital (Hines, Ill.) from Jan. 1, 1931, to Jan. 1, 1945. Of the patients studied, 70 per cent complained of pain and hematuria as the initial symptoms, and 31 per cent had hematuria only. Delay in investigation of these early symptoms was responsible for late diagnosis in many cases, but 11 per cent of the patients had no symptoms at all.

A palpable abdominal mass was present in 41 per cent of the series at the time of initial examination, while 33 per cent had blood in the urine. Intravenous urography was diagnostic in only 16 per cent of cases in which it was done, but retrograde pyelography was diagnostic in 66 per cent.

At the original examination, 36 per cent were found to have metastases, and at the time of operation many others were found to be inoperable because of local extension or metastatic spread. Only 35 per cent of the patients finally had the tumor and kidney removed, and over 50 per cent of these were known to be dead in one year. In inoperable cases receiving x-ray therapy, there was no striking prolongation of life. One-third of 15 cases receiving preoperative therapy showed some reduction in the size of the tumors, but there was delay in healing of the operative wounds.

The group of patients reported includes 86 per cent with tumors of the parenchymal portion of the kidney and 14 per cent in which the tumors were of pelvic or caliceal origin.

The author makes a plea to the profession for adequate diagnostic study in any patient complaining of

hematuria, and for the education of the public as to the importance of this symptom.

JOHN O. LAFFERTY, M.D.

**Nephrocalcinosis.** John H. Vaughan, Merrill C. Sosman, and Thomas D. Kinney. *Am. J. Roentgenol.* 58: 33-45, July 1947.

In nephrocalcinosis there are calcium deposits in the renal parenchyma—in the pyramids alone or in the cortex, or both. Pathological calcification in human tissue may result from (1) abnormally high concentrations of calcium or phosphate in healthy tissues or (2) from local tissue damage with precipitation of calcium in the presence of normal blood levels. The areas of predilection are those where there are marked changes in the pH of the media or where there is an increased concentration of phosphatase. Calcification of the first type occurs occasionally in hyperparathyroidism, with multiple deposits in the kidneys as well as throughout the body, particularly in the peritubular areas, the lungs, the gastric mucosa, and the large blood vessels. It has been observed following intoxication with vitamin D. The second type of calcification is found in those diseases of the kidneys in which the renal tissue is necrotic, severely damaged, or chronically inflamed. Renal calculus formation is not the rule.

A case of nephrocalcinosis is reported in detail. A 26-year-old man, six years prior to being admitted to the hospital, had suffered from a streptococcal sore throat followed by what appeared to have been a typical acute glomerulonephritis. A high milk diet was followed for six years. Symptoms of renal insufficiency developed and a diffuse extensive calcification of both kidneys was found on roentgen examination. Two years later a terminal uremia developed, a course similar to that commonly seen in chronic glomerulonephritis. The presence of calcium in the kidney was thought to be no more than a contributing factor in the production of the renal insufficiency. Bones of the body showed none of the changes one would associate with hyperparathyroidism. There was no resemblance to tuberculous calcification of the kidneys. The appearance of the kidneys was unique and could be attributed only to some disease or injury uniformly involving all of the renal tissue. Normal blood calcium and phosphorus levels were present when the patient was first seen. It is suggested that early in the disease, when there was still active inflammation in the renal parenchyma, the patient consumed enormous quantities of milk, so that large amounts of calcium were excreted in the urine. At this time there was active degeneration of renal tissue creating a medium in which precipitation would be likely to take place. The acute nephritis subsided into a latent or subacute phase, which eventually was responsible for such scarring that a state of renal insufficiency resulted. Laying down of calcium in the damaged renal tissue occurred in the presence of long-continued urinary concentration of calcium. Calcium deposition in the kidneys was not the prime factor in the renal failure.

CLARENCE E. WEAVER, M.D.

#### THE BLOOD VESSELS

**Phlebography for the Study of Obstruction of the Veins of the Superior Vena Caval System.** Sol Katz, Hugh Hudson Hussey, and James Ross Veal. *Am. J. M. Sc.* 214: 7-22, July 1947.

Phlebography offers a method for defining and lo-

calizing lesions of the veins and determining the distribution of collateral circulation. For lesions of the superior vena cava the external jugular vein is preferred as the site for injection. In lesions of the subclavian, axillary or brachial veins, the median basilic is the site of choice. Thorotrast or 35 per cent or 70 per cent diodrast is used. In order to visualize the superior vena cava or innominate vein, 30 c.c. of the medium is injected rapidly. In all other instances 15 c.c. is used and speed of the injection is less important.

In the superior vena cava, the main causes of obstruction have been, in order of frequency, aortic aneurysm, bronchogenic carcinoma, and mediastinal lymphoma. Less frequent causes are metastatic cancer of the mediastinal lymph nodes, superior vena cava thrombosis, and mediastinitis. The location and extent of the collateral circulation depend on the site, duration, and completeness of the obstruction. With obstruction above the entrance of the azygos vein, this vein and its tributaries are the main collateral channels, and therefore the visible collateral circulation is not extensive. When the obstruction is below the azygos vein, an extensive collateral circulation is visible on the chest and abdomen.

The etiology of obstruction of the innominate vein is the same as for the superior vena cava, but thrombosis is more frequent. The left innominate vein is more often obstructed by aneurysm because of the intimate relationship of this vein to the aortic arch. When an innominate vein is obstructed, the blood finds its way back to the heart through the other innominate. The collateral veins traverse the midline and include all the tributaries of the innominate, jugular and subclavian veins.

Subclavian and axillary vein obstruction is commonly due to thrombosis, and, in the case of the subclavian, is usually complete. In order of frequency, the causes of thrombosis are metastatic carcinoma, congestive heart

failure, trauma or effort, and constriction by scar tissue. Incomplete obstruction of the axillary vein is a frequent sequel to radical mastectomy, and has been shown to be due to angulation of the vein at the axilla. When the first part of the axillary vein is occluded, the cephalic vein is the main collateral vessel. When the entire axillary vein is occluded, the collaterals develop from the minor branches of the basilic, brachial, and cephalic veins to communicate with small veins of the neck and thorax. When the axillary and subclavian veins are both obstructed, the collateral circulation is essentially the same as with occlusion of the axillary vein alone. The collateral network is more extensive however.

Phlebography is simple, and, with diodrast, almost entirely safe. It provides anatomic details which could otherwise be obtained only by dissection. There is no other means of differentiating clinically obstruction of both innominate veins from superior vena cava obstruction or axillary vein thrombosis from subclavian obstruction.

BENJAMIN COPELAND, M.D.

## TECHNIC

**A Radiopaque-Plastic Injection Mass.** Charles E. Tobin. *Anat. Rec.* 98: 137-145, June 1947.

Röntgenograms as well as corrosion preparations were made from infant and adult specimens injected with a radiopaque plastic consisting of 10 per cent iodoform and 90 per cent vinylite (12.5 per cent vinyl resin in acetone stained with fast dyes). Such roentgenograms are useful for diagnostic, teaching, and research purposes. The hard casts formed by the mass in the injected areas will withstand clearing, bacterial maceration, and chemical corrosion. The casts can be retained in their relationship to the bony structures by weak alkaline corrosion or bacterial maceration of the soft tissues.

## RADIOTHERAPY

**Treatment of Cancer of the Face, Mouth, and Neck with Irradiation.** Charles L. Martin and Carleton Wright. *J. A. M. A.* 134: 861-866, July 5, 1947.

A description of the authors' program for treating cancer of the face, mouth, and neck by irradiation and electrosurgery is given with a discussion of the types of radiation and the technics of application used for lesions of the various sites. Reproductions of photographs demonstrate lesions before and following treatment. Tables showing the results of irradiation in 119 patients with proved cancer of the face and lip; of 113 patients with carcinoma of the mouth, and of 138 patients with palpable cervical lymph nodes which were considered to be neoplastic are presented and show an absolute three-year cure rate of approximately 25 per cent. The authors make a plea for an "all-out effort to produce a cure" with the first series of treatments—a precept which cannot be too strongly emphasized.

R. S. MACINTYRE, M.D.  
(University of Michigan)

**Malignant Goiter. Lessons to be Learned from a 20-Year Follow-Up.** Robertson Ward. *West J. Surg.* 55: 383-388, July 1947.

This author relates the lessons he has learned in

twenty years of study of malignant goiter. Conclusions based on a five-year follow-up are quite inadequate when dealing with thyroid carcinoma. Inordinately long survival of the patient with apparently dormant malignant tissue is common, and eight- to ten-year remission after operation alone or with radiation is not unusual. This is particularly true of tumors of lateral origin or those in which lateral metastases are the presenting symptom.

The problem of thyroid carcinoma is inseparable from the problem of nodular goiter. Malignant change associated with true exophthalmic goiter is rare in the author's experience. Nodular goiter, on the other hand, is the breeding ground of malignancy and almost all carcinomas of the thyroid amenable to treatment grow in nodular form. Of 96 cases of solitary adenoma of the non-toxic variety, 15.6 per cent were found to be definitely malignant and an additional 22 per cent to be benign neoplasms. Cole, in a similar series of 92 cases, found 24 per cent to be malignant (*J. A. M. A.* 127: 883, 1945). The only safe procedure, therefore, in this condition is complete lobectomy. Surgery is also indicated for all nodular goiters in adult males and in children, because of the high incidence of malignant change in these two groups. Recent growth or the de-

velopment of pressure symptoms in patients with longstanding goiter is always an indication for surgery.

Lateral aberrant thyroid nodules are considered by some as metastases from primary neoplasm of the homolateral lobe of the thyroid, while others believe that they are fetal rests having low or potential malignancy. In the author's experience, 13 of 15 such cases were either definitely malignant or had distinct malignant potentialities and he believes all such nodules should be removed by block dissection, including the homolateral lobe of the thyroid.

The author's experience regarding postoperative radiation therapy indicates that tumors predominantly of the papillary type respond well to irradiation, either by becoming completely arrested or remaining dormant for long periods. Since adequate irradiation entails no little discomfort and morbidity, he hesitates to advise it in those tumors found pathologically to be completely confined by the capsule of the adenoma of origin. In many of those cases, radiation therapy has been withheld pending evidence of recurrence or has been used only on distant metastases. Despite the fact that undifferentiated and diffuse carcinomas are not cured or even temporarily arrested by irradiation, many lives have been prolonged and apparent cures obtained by this method after recurrence has taken place in patients with tumors of the papillary type. Instances have been observed in which irradiation alone has controlled growth and maintained patients in reasonably good health for as long as eleven years after postoperative recurrence. The author believes that roentgen therapy should be given postoperatively in all cases in which there is reasonable doubt concerning complete removal of neoplastic tissue, but that it is likely to be effective only in those tumors of predominantly papillary type.

Locally recurrent nodules should be removed surgically. Many are tumor thrombi in the proximal stumps of veins. They are radioresistant because of the surrounding venous plexus and because the malignant adenoma which spreads by vein is usually not of the papillary type.

B. S. KALAYJIAN, M.D.

**The Object, the Value, and the Technique of Preoperative and Postoperative X-Ray Treatment in Carcinoma of the Breast.** George E. Pfahler and George P. Keefer. *Surg., Gynec. & Obst.* 85: 35-46, July 1947.

The authors confine their discussion of preoperative and postoperative roentgen therapy of carcinoma of the breast to stage 2 cases with axillary metastases proved by operation. They agree with Rödén (*Acta radiol. Suppl. LVII*, 1945) that "sinus catarrh" is a "preliminary stage to the metastasis." They accept the criteria of operability outlined by Haagensen and Stout (*Ann. Surg.* 118: 1032, 1943).

Preoperative irradiation should be given whenever there are palpable axillary lymph nodes, since, in general, cases with axillary metastases are apt to be more malignant histologically and therefore more radioresistant. Though figures to prove the value of preoperative therapy are not available, the authors believe that it is indicated in view of the fact that irradiation devitalizes the more malignant cells and normal tissues are made less receptive to implantation, as has been shown experimentally.

The authors allow a period of about two weeks for

preoperative irradiation, giving approximately 1,000 to 1,400 r to the tumor area, through each side of the breast and through the axillary and suprasternal region. The rays are projected tangentially so as to miss the lung. Axillary and supraclavicular portals are also used, and occasionally a posterior axillary port. Factors are 180 to 200 kv., 50 cm. distance, 0.5 mm. copper filtration. Operation should follow in a few days or a week, as delay permits growth of radioresistant cells and extension of the malignant process.

Postoperative therapy is begun ten days to two weeks following surgery. A posterior axillary portal is used, because the arm generally cannot be raised. A large portal covers the supraclavicular and anterior axillary regions. A third large portal covers the chest and extends (for the left breast) from the right border of the sternum to the left anterior axillary fold. Smaller doses are used over this portal, with 135 kv. and 2 mm. Al filtration, and the aim is to deliver to this region a total of 1,800 to 2,400 r, counting both preoperative and postoperative treatment. A similar dose is given to the axilla, coracoid, and supraclavicular region.

Ovarian sterilization is recommended "for all women with cancer of the breast who are still in the menstrual time of life."

The authors review the theoretical value of postoperative therapy in destroying any remaining growth and rendering the fields less favorable for the growth of cancer.

Statistical records show a great variation in terminal results following treatment by surgery alone and surgery and irradiation combined. A review of the figures from several large clinics seems to indicate that the average five-year survival rate for stage 2 cases is 27.4 per cent for surgery alone and 37.6 per cent for surgery followed by roentgen therapy.

JOHN A. COCKE, M.D.

**Treatment of Cancer of the Breast.** Leonardo Guzman. *Cancer (Santiago de Chile)* 5: 178-200, 1945.

This is a very interesting collection of opinions on the treatment of carcinoma of the breast by outstanding specialists in the field of cancer in answer to a questionnaire circulated by the author. Dr. Guzman finds that there is no unanimity of opinion in so far as preoperative radiotherapy is concerned. He himself uses it in cases in which there is retraction of the skin or fixation of the axillary metastases. He feels that to acquire a logical criterion for the treatment of carcinoma of the breast, it is necessary to classify these tumors according to their clinical behavior, degree of extension, and histologic appearance.

JUAN A. DEL REGATO, M.D.

**Treatment of Cancer of the Uterine Cervix.** Leonardo Guzman. *Cancer (Santiago de Chile)* 5: 79-93, 1945.

The well known South American radiotherapist, Dr. Guzman, has written a very comprehensive monograph on the treatment of carcinoma of the cervix for the benefit of the general practitioners. He emphasizes that cancer of the cervix is not a disease of old women, since 35 per cent of his patients were under the age of forty years. In respect to the pathologic grading, he is of the opinion that anaplastic tumors may have a more favorable prognosis in the earlier stages while, on the contrary, they have a worse prognosis in the later stages.

JUAN A. DEL REGATO, M.D.

**Nitrogen Mustards in Hodgkin's Disease. Report on 21 Cases and 4 of Other Reticuloses.** M. I. R. Ap-Thomas and H. Cullumbine. *Lancet* 1: 899-901, June 28, 1947.

Nitrogen mustards have been used by the authors in 25 patients, 21 with Hodgkin's disease in an advanced stage, some recurrent after radiotherapy, and 4 with other reticulososes. The diagnosis in all cases was confirmed by biopsy. Sixteen patients were treated with methyl-bis (beta-chloro-ethyl) amine hydrochloride ("bis" form) and 5 with tris (beta-chloro-ethyl) amine hydrochloride ("tris" form). Nausea and vomiting occurred at some period in all cases except one. Thrombosis at the site of injection occurred in 6 patients, 2 of whom received the tris form. All 21 patients with Hodgkin's disease improved after the first course, but many of them required further treatment for recurrence of symptoms. Of the 13 patients who had two courses, 12 showed improvement after the second course; of the 4 who had three courses, only 2 were improved. It is possible, therefore, that the disease responds readily at first but then gradually becomes more resistant to the drug. In the authors' experience no better results have been obtained with the tris form than with the bis compound.

On comparing the results obtained with nitrogen mustards with those with deep x-ray therapy at the Holt Radium Institute, it was found that improvement in general condition, nodes, and other signs was produced more quickly with nitrogen mustard than with x-rays but was of shorter duration. Vomiting is more likely and more severe after nitrogen mustard than after radiotherapy. There is no skin or mucosal reaction with intravenous nitrogen mustard, but local thrombosis at the injection site occurred in a few cases. Leukopenia is certain and sometimes severe after nitrogen mustard, whereas after radiotherapy it occurs only when extensive areas are treated and can be controlled by stopping treatment.

In the authors' opinion radiotherapy is to be preferred as a first treatment in Hodgkin's disease. The possibility of combining radiotherapy with nitrogen mustard therapy is being investigated.

Of 2 patients with lymphosarcoma treated with nitrogen mustard, one showed improvement; one case of reticulum-cell sarcoma and one of mycosis fungoides showed no improvement.

#### RADIOACTIVE ISOTOPES

**Radioactive Phosphorus as an External Therapeutic Agent in Basal Cell Carcinoma, Warts and Hemangioma.** Bertram V. A. Low-Beer. *Am. J. Roentgenol.* 58: 4-9, July 1947.

The discovery of artificial radioactivity has made available substances which radiate beta particles only. Radioactive phosphorus ( $P^{32}$ ) is one such substance. It loses one-half of its initial activity in 14.3 days. Absorption measurements have shown that approximately 48 per cent of the radiation is absorbed in the first millimeter of water or tissue.

The radioactive phosphorus used by the author in the treatment of superficial skin lesions was an aqueous solution of disodium hydrogen phosphate containing 15 mg. of the salt per cubic centimeter of water. Office type blotting paper 0.4 mm. thick and weighing 21 mg. per square centimeter was cut to size to cover the lesion and to allow a safety margin of 0.3 to 1.0 cm., depending on the type of lesion. The blotting paper was

backed by some kind of adhesive tape and placed on a good drying surface of low heat. A measured amount of  $P^{32}$  solution was then soaked into the blotting paper. After drying, it was applied over the skin lesion and secured in place. It was found that the minimum exposure which produces a faint but discernible "threshold" erythema is 34 microcurie-hours per square centimeter.

Three hundred and one skin lesions were treated, including basal-cell carcinoma, hyperkeratoses, verruca, plantar warts, subungual warts, and hemangioma. Tables of dosage are given as well as results of treatment. It is concluded that artificially radioactivated phosphorus, as a pure beta radiator, can be used satisfactorily in the treatment of superficial skin diseases, though there is no intention to advance the method in competition with other well established modes of treatment.

CLARENCE E. WEAVER, M.D.

**Retention of Radioactive Iodine in Thyroid Carcinomas. Histopathologic and Radio-Autographic Studies.** L. D. Marinelli, F. W. Foote, R. F. Hill, and A. F. Hocker. *Am. J. Roentgenol.* 58: 17-30, July 1947.

Tissue sections and radio-autographs from 19 cases of thyroid carcinoma were prepared and studied. The patients were given tracer or therapeutic doses of radioactive iodine as sodium iodide in water solution by mouth. Twenty-four to forty-eight hours later, blocks of tissue from the tumor were removed. The method of preparation of the tissue is described in detail. Frozen sections were not as satisfactory as those prepared with the paraffin technic. Some exposures of photographic plates or films to the tissue were as long as three weeks. Radioactive material was deposited only in epithelium and colloid; none in connective tissues.

A discussion is given of the various types of thyroid carcinomas and their histopathologic features, and photomicrographs are reproduced with radio-autographs alongside to illustrate the pick-up pattern of radioactive material in the various tumors. From the studies it is clear that certain types of thyroid carcinoma do possess the ability to accumulate radioactive iodine. It appears that structural type is an important determining factor. Generally speaking, pick-up of the radioactive material is closely linked with structural qualities which include orderly cell arrangement in follicular pattern and the presence of colloid-like material. In the 19 cases in this study there were 10 in which the presence of radioactive material was detectable by means of radio-autographic determination. Five of these were examples of benign metastasizing struma. The remaining 5 had the structure of follicular adenocarcinoma in some portion of the material studied. Approximately 15 per cent of thyroid cancers may be expected to accumulate radioactive iodine in some degree. The most favorable histopathologic type of thyroid cancer is the so-called benign metastasizing struma.

As to therapy, a very conservative attitude would point to the treatment of metastasizing struma only. The hopelessness of metastatic thyroid cancer and the radioresistance of the more functional forms, however, counsel a more extended therapeutic trial in all cases in which external measurements reveal the presence of the isotope in metastases irrespective of the possible heterogeneous deposition of iodine that may be present therein. Therapeutic means, too, may be developed later to increase the deposition of radioiodine in thyroid tumors.

CLARENCE E. WEAVER, M.D.

## RADIATION EFFECTS

**Radiation Doses Received by the Skin of a Patient During Routine Diagnostic X-Ray Examinations.** J. H. Martin. *Brit. J. Radiol.* 20: 279-283, July 1947.

By ionization chamber measurements in air and on phantoms, the skin dose received in routine diagnostic examinations was determined. In some instances large doses were administered. These can be reduced by increasing the target-skin distance and by increasing the filtration. Increasing filtration to 1.0 mm. of Al is recommended.

The doses received by the skin in some typical examples are chest 0.04 to 0.4 r per exposure; intravenous pyelography, 0.5 to 1.5 r per exposure; skull and sinuses, 4 r per film for anteroposterior and 1.0-2.0 r for lateral views; spine, 2 to 34 r per film; dental films 1 to 15 r per exposure; pregnancy, average 24 r per film (highest 65 r); screening, between 10 and 20 r per minute in the usual case.

SYDNEY J. HAWLEY, M.D.

**Effect of Irradiation, Immunity and Other Factors on Vaccinal Infection. A Review Illustrated by the Report of a Secondary Ocular Infection Treated with Roentgen Rays.** Hal W. Pittman, Lawrence Byerly Holt, and George T. Harrell. *Arch. Int. Med.* 80: 61-67, July 1947.

An accidental human ocular infection with vaccinia virus, secondary to inoculation of the arm for smallpox, was treated with roentgen irradiation, 220 r in air in three days (22 r per minute for four minutes on the first day and for three minutes each succeeding day), without the development of residual scarring. Vaccinal infection occurs when active virus meets a susceptible cell and becomes established intracellularly. It may be prevented or treated by measures designed (1) to inactivate the virus, (2) to alter the host cell, and (3) to hinder the spread of the virus through tissue. The mechanism by which roentgen therapy may act is not clearly known.

**Effect of Large Dosages of Irradiation on Gastric Acidity.** Irving B. Brick. *New England J. Med.* 237: 48-51, July 10, 1947.

This is a comparative study of three different groups of patients all of whom received irradiation for cancer. The irradiation factors were 1,000 kv., 3 ma., 3 mm. tungsten filter, and 70 or 100 cm. focal distance. The ports measured 10 X 10 cm., and the field irradiated usually involved the antrum of the stomach. The object of the study was to determine the effect of the irradiation on the acid-secreting factors in the stomach. None of the patients was known to have had any stomach abnormality prior to the irradiation.

The first group consisted of 4 patients in whom gastric resection was done. Three of these had perforation of the stomach from an ulcer; one of the 3 had a low normal gastric acidity, another a high normal, and a third a normal gastric acidity. Hemorrhage occurred in 2 patients, in one of whom the gastric acidity was high and in the other low. The total amount of radiation received by these patients was between 5,000 and 6,000 r. There did not seem to be any depression of the acid-secreting function of the stomach.

The second group consisted of 5 patients who defi-

nately had ulcer of the stomach but improved without operation. The amount of radiation varied from 2,500 to 5,000 r. In none of these patients was there evidence of depression of the acid-secreting function of the stomach.

The third group consisted of 5 patients with normal x-ray findings but symptomatic changes suggestive of ulcer. The average dose was 5,000 r but no definite relationship could be demonstrated between the dose and the gastric acidity. The mean acidity values were slightly higher than in the previous groups.

From the study of these patients, the author believes that there is no place for irradiation treatment in peptic ulcers since so many patients exposed to irradiation developed ulcers or ulcer symptoms and no change in the acidity of the gastric contents is definitely demonstrated.

JOHN B. McANENY, M.D.

**EDITOR'S NOTE:** In a letter to the *New England Journal of Medicine* (237: 566, Oct. 9, 1947) Dr. Walter L. Palmer criticizes Dr. Brick's conclusions on the ground that the radiation was delivered to the antrum of the stomach, while the acid-secreting cells are located in the middle and upper portions. He also objects to the "sweeping statement . . . that 'radiation has no place in the treatment of peptic ulcer, since it has been shown that deleterious effects on the stomach can be obtained with this agent'." The fact that radiation may produce tissue necrosis under certain circumstances (and to this Palmer attributes the ulcers developing in the cases described) does not prove radiation to be of no value under other circumstances.

In replying to Dr. Palmer in the same issue of the journal, Dr. Brick states that while it is true that the chief radiation effect in his cases was upon the antrum and pylorus, at operation there was evidence of an effect throughout the stomach, in the form of edematous thickening and vascularity. As particularly relevant to the discussion he cites a study of Palmer and Templeton (*J. A. M. A.* 112: 1429, 1939) which showed an extreme variability of the reaction of gastric acidity to radiation. It is this variability in the effect of radiation on gastric acidity as well as in the radiation injury to the stomach that deserves emphasis. With the dosages used both by Palmer and Templeton and by the author the effect on acidity was unpredictable in the individual case and the possibility of radiation injury was present. "I believe," says Brick, "that with the present knowledge of radiation effect on the stomach with and without peptic ulcer, the use of radiation in the treatment of peptic ulcer should be limited to investigative personnel doing carefully controlled clinical research, as illustrated notably by Dr. Palmer and associates. To date, no one has claimed to be able to produce destruction of the acid-secreting cells of the gastric mucosa with radiation, regardless of dosage."

**Surgery in Radiation Injury of the Stomach.** Ralph F. Bowers and Irving B. Brick. *Surgery* 22: 20-40, July 1947.

Six cases of radiation injury of the stomach treated by surgery are reported. All followed x-ray therapy to the posterior abdominal structures for metastases from testicular tumors. The depth dose received in these cases, calculated at the level of the eleventh dorsal vertebra, ranged from 4,800 r in fifty-three days to

6,456 r in thirty-two days (1,000 kv., 3 ma., 3 mm. tungsten filter, and a focal skin distance of 70 or 100 cm., 88 to 40 r, respectively being delivered per minute). Many patients receiving 5,000 r or more to the epigastric region at this level show no gastro-intestinal symptoms; on the other hand, ulceration of the stomach may occur following much smaller doses.

Some patients with radiation ulcers improve temporarily under medical care. Indications for surgery are the same as for ordinary peptic ulcer, namely, (1) perforation, (2) hemorrhage, (3) intractability of symptoms; on the other hand, ulceration of the stomach may occur following much smaller doses. Complications from these ulcers are more frequent than from the usual peptic ulcer. The skin and abdominal wall are relatively free of radiation change. Grossly the gastric and intestinal lesions have an edematous, thickened, whitish appearance and are firmer to palpation than the normal bowel wall. There was some increase in vascularity in all cases; two cases showed a generalized radiation effect without large ulcers and it is believed that small ulcers had healed before operation. Difficulty was encountered in delineating the limits of the radiation injury in planning resection and anastomosis. Three of the ulcers in the antral portion of the stomach had perforated; hemorrhage occurred in 3 cases, one of which had also perforated; one was an uncomplicated pyloric ulcer. In one case a fistula developed probably due to failure of the duodenal stump to heal as a result of radiation injury. Operative treatment consisted in gastric resection with anastomosis to the jejunum.

The authors feel that operative intervention is indicated as a life-saving measure in the presence of hemorrhage. They also believe that surgical resection should not be too long delayed, because of the progressive nature of radiation injury. Operative intervention in these cases was undertaken from ninety-five to two hundred twenty-three days after completion of irradiation. Symptoms attributable to gastric damage began from one to four months following completion of irradiation.

Operative findings in the 6 cases were as follows: (1) perforation of a 3-cm. ulcer on the posterior wall of the antrum; (2) thickened mucosa, submucosal ecchymosis, and generalized petechial hemorrhage of gastric mucosa; (3) ulcer 2 cm. in diameter and 2 mm. deep proximal to the pyloric ring, with mucosal petechial hemorrhages; (4) perforated ulcer 2 cm. in diameter on the posterior antral wall; (5) ulcer 1.5 cm. in diameter on the posterior wall near the pyloric ring; (6) subacute perforation of a 3 X 3 X 2 cm. ulcer in the gastric antrum with peritonitis.

The great individual variation of the gastric and intestinal walls in their response to irradiation is emphasized.

J. E. WHITELEATHER, M.D.

**Endothelial-Cell Sarcoma of Liver Following Thorotrast Injections.** H. Edward MacMahon, Albert S. Murphy, and Margaret I. Bates. *Am. J. Path.* 23: 585-611, July 1947.

A 58-year-old woman was given thorotrast for the visualization of the liver. With the aid of this diagnostic procedure, combined with positive serologic tests, it was possible to make an accurate diagnosis of hepatic syphilis with gumma. Following specific therapy, the patient made a clinical recovery and for twelve years lived a reasonably normal life. At the age of seventy, she died suddenly. Autopsy findings con-

firmed the diagnosis of syphilis and in addition revealed a primary hemorrhagic endothelial-cell sarcoma of the liver, the source of fatal hemorrhage, and very widespread irradiation damage, affecting particularly the liver and hematopoietic system. Evidence is produced from a study of this case to support the debatable contention that thorotrast, in sufficient quantities, as a radioactive substance, is injurious. Evidence is also presented to show that thorotrast, like other radioactive substances, in sufficient time may act as a sarcogenic agent.

**Certain Aspects of the Action of Radiation on Living Cells.** F. G. Spear (editor). *Brit. J. Radiol., Supplement No. 1*, 1947.

The British Journal of Radiology has published in the form of a Supplement the Report of the London Conference arranged by the British Institute of Radiology to consider the action of radiation on living cells.

The introductory paper by L. H. Gray deals with the distribution of the ions resulting from the irradiation of living cells by beta and gamma rays, x-rays, neutrons, and alpha particles. The remaining papers constitute four groups, taking up the effects of radiation, respectively, on viruses, dilute aqueous solutions, chromosomes, and germ cells with special reference to man.

With the supplement comes a four-page glossary of biological, physical, and chemical terms which should be valuable to those less familiar with experimental studies of the type presented here.

**Radiosensitivity of Erythroblasts.** Margaret A. Bloom and William Bloom. *J. Lab. & Clin. Med.* 32: 654-659, June 1947.

This is a further report on the work of the Plutonium Project which was described by Wm. Bloom in the September 1947 issue of *RADIOLOGY*. The present paper deals particularly with the effect of external irradiation on the erythroblasts. The agents used externally were x-rays and fast and slow neutrons. The sources of internal radiation were beta and gamma emitters, including sodium<sup>24</sup>, barium<sup>130</sup>, lanthanum<sup>140</sup>, strontium<sup>90</sup>, and phosphorus<sup>32</sup>, injected intraperitoneally, and yttrium<sup>91</sup> injected intravenously. Alpha emitters were also tried, in the form of plutonium nitrate or citrate. Radium from which the radon had been blown off was also given intraperitoneally. Rats, mice, rabbits, and chickens were used for the experiments.

It is apparent that the amount of destruction of the cells in the bone marrow is proportionate to the dose of radiation no matter how it is given. In all cases, erythropoietic cells proved to be more radiosensitive than myelocytopoietic cells, and these in turn than the megakaryocytes. Large numbers of degenerating erythroblasts in mammalian bone marrow after irradiation had the appearance of degenerating lymphocytes, and it is believed that the two types of cells may have been confused in the past and that thus the radiosensitivity of the erythroblasts was not appreciated. It is further pointed out that the radiosensitivity of erythroblasts may often have been overlooked because of the acute changes in the leukocytes (especially lymphocytes) in the peripheral blood in contrast to the minor changes in the more long-lived erythrocytes.

SYDNEY F. THOMAS, M.D.

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